Archives of Neurology and Psychiatry

VOLUME 19 APRIL, 1928 NUMBER 4

THE FOREBRAIN

A STUDY AND RECONSTRUCTION BASED ON THE METHOD OUTLINED BY THE AUTHORS *

ADOLF MEYER, M.D.
BALTIMORE
AND
LOUIS HAUSMAN, M.D.
NEW YORK

The study of the structure of the central nervous system has been approached in various ways. The method that forms the basis of this presentation was described in a previous paper,[1] in which the segmental and suprasegmental systems of the hindbrain and midbrain were fully discussed and illustrated. The method takes into account the sequence of development which the data and methods of embryology and comparative anatomy reveal. By combining this perspective with a survey of the facts of neurophysiology and of experimental and pathologic neuroanatomy, a dynamic approach to the study of the central nervous system is provided.[2]

It is our purpose to present the mechanisms of the forebrain in terms of this analysis, with the various systems translated into three dimensions by means of an accurate plasteline reconstruction, made from a complete series of Weigert-stained cross-sections of a human adult brain.

The apparatus of the forebrain, in its ultimate development, presents one of the most plastic outfits of the neural tube in higher animals. The evolution of its pattern throughout the vertebrate phylum reflects the various environmental adaptations the animal has made and can make. It is recognized at a very early stage of development as the end-bud of the primitive neural tube, evolving over the olfactory and the optic segmetnal structures and harboring the remarkable endocrine organs, the hypophysis and the epiphysis.

---

* Read at the Fifty-Third Annual Meeting of the American Neurological Association, Atlantic City, N. J., May, 1927.

1. Meyer, Adolf; and Hausman, Louis: A Reconstruction Course in the Functional Anatomy of the Nervous System, Arch. Neurol. & Psychiat. **7**:287 (March) 1922.

2. This method has been adapted for teaching purposes and is now in use as an elective course at the Johns Hopkins Medical School and as a prescribed course for first year students at the Cornell Medical School.

We recognize the median division of the forebrain (or thalamencephalon) with its cavity (or third ventricle) opening through the foramen of Monro on either side into the cavity of the lateral division of the forebrain (or cerebral hemisphere), with its olfactory protrusion at the base. The corpus striatum with its connections develops over the olfactory structures, and ultimately, crowding back the dorsal choroid membrane, the cerebral cortex with its systems of communication develops.

The visualization of the forebrain in terms of structures of the midline, lateral wall and hemisphere provides a convenient approach to the reconstruction of the forebrain.

Fig. 1.—A sagittal section through the midline of an adult human brain for orienting the midline structures of the forebrain. (Pal-Weigert stain.)

STRUCTURES OF THE FOREBRAIN

The Midline Structures.—From the midbrain forward, the dorsal median line of the neural tube (figs. 1 and 2) consists of: (1) the posterior commissure (beneath which the aqueduct of the midbrain deepens into the cavity of the median forebrain or third ventricle); (2) the pineal recess and body; (3) the superior or habenular commissure; (4) the roof membrane of the "third ventricle" spreading between the tenia edges of the thalami (or epithalami) and extending posteriorly as the epipineal pocket and anteriorly as far forward as (5) the neuropore region (the most anterior end of the fetal dorsal suture and the last part of the neural tube to close in the embryo). These structures comprise the dorsal median line or roof of the ventricle of the median forebrain.

The anterior median line or lamina terminalis, which forms the anterior wall of the "third ventricle," represents the terminal slope of the neural tube (fig. 1). It extends between the neuropore (above the level of the anterior commissure) and the optic chiasm. The upper or dorsal limit of the anterior wall is marked by the anterior commissure from which the lamina terminalis slopes down to form the preoptic recess at its junction with the optic chiasm; from there the ventral median line extends back to the midbrain as the floor of the ventricle, presenting: (1) the postoptic recess, (2) the infundibulum meeting the hypophyseal gland growing from the pharynx, (3) the tuber cinereum, (4) the mammillary region and (5) the interpeduncular ganglion pushed backward over the ventral or effector plate of the midbrain (fig. 1).

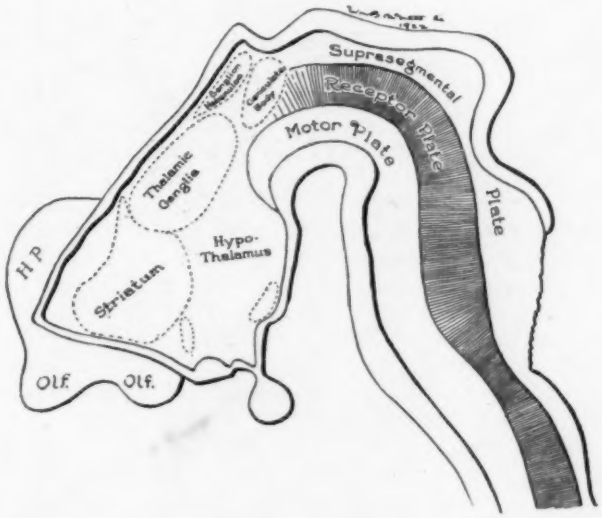


Fig. 2.—Median section through the brain of a human embryo (after a His model). In addition to the midline structures, the lateral wall of the cavity of the median forebrain (or third ventricle) and the hemisphere pocket are shown. *H. P.* indicates the right hemisphere pocket or cerebral hemisphere; *Olf.*, olfactory base.

The Lateral Walls of the Ventricle of the Median Forebrain.—A median section through the brain of a human embryo shows the following structures of the lateral wall (fig. 2): (1) The thalamus mass (consisting of hypothalamus, thalamus proper or mesothalamus and epithalamus); (2) anteriorly, two protrusions: (a) the optic vesicle near the ventral part of the lamina terminalis, and (b) the olfactory and hemisphere protrusion projecting as a pocket from the dorsal part of the lamina terminalis.

The following abbreviations apply to figures 3 to 15 inclusive: *Ac. N.*, acoustic nerve; *Amyg.*, amygdaloid nucleus; *Ant. C.*, anterior commissure; *Ant. C. Olf.*, olfactory portion of anterior commissure; *Ant. C. T.*, tail of anterior commissure; *Caud. H.*, head of caudate nucleus; *Caud T.*, caudate tail; *Caud. Tub.*, caudate tubercle; *C. C. B.*, body of corpus callosum; *C. C. G.*, genus of corpus callosum; *C. C. S.*, splenium of corpus callosum; *Ch. Pl.*, choroid plexus; *C. I. A.*, space for anterior part of internal capsule; *C. I. P.*, space for posterior part of internal capsule; *C. I. R. L.*, space for retrolenticular part of internal capsule; *C. I. S. L.*, space for sublenticular part of internal capsule; *C. Ma.*, mammillary body; *Cornu A.*, cornu ammonis; *Corp. L.*, corpus Luysii or hypothalamic nucleus; *Epith.*, epithalamus; *Ext. G.*, external geniculate ganglion; *Ext. G. O. N.*, external geniculate ganglion, with portion of optic nerve entering it; *F. F.*, Forel's field; *Floc.*, flocculus; *F. M.*, foramen of Monro; *F. N.*, facial nerve; *For. B.*, fornix body; *For. C.*, fornix column; *G. D.*, dentate gyrus; *G. Hab.*, ganglion habenulae; *H. C.*, hippocampal commissure; *Hypoth.*, hypothalamus; *Inf.*, infundibulum; *Inf. C.*, inferior colliculus; *Inf. O.*, inferior olive; *Int. G.*, interpeduncular ganglion; *I. O. Dec.*, interolivary decussation; *Lat. F.*, lateral fillet; *Lat. O. Tr.*, lateral olfactory tract; *Lam. T.*, lamina terminalis; *Ma. Th. B.*, mammillothalamic bundle; *Mes. Olf. Tr.*, mesial olfactory tract; *M. P.*, motor or effector plate; *Oc. N.*, oculomotor nerve; *Olf. Tub.*, olfactory tubercle; *Opt. Ch.*, optic chiasm; *Opt. N.*, optic nerve; *Opt. Tr.*, optic tract; *P. Com.*, posterior commissure; *Pi. B.*, pineal body; *Pi. Rec.*, pineal recess; *P. L. B.*, posterior longitudinal bundle; *Post. O. Rec.*, postoptic recess; *Pre. O. Rec.*, preoptic recess; *Pulv.*, pulvinar; *Putam.*, putamen; *Red. Nuc.*, red nucleus; *Ret. B. M.*, retroflex bundle of Meynert; *R. Tr.*, rubrospinal tract; *R. Tr. Dec.*, decussation of rubrospinal tracts; *S. Cbll. A. 1.*, the superior cerebellar arm before decussation; *S. Cbll. A. 2.*, superior cerebellar arm after decussation; *S. Cbll. A. Dec.*, decussation of superior cerebellar arms; *S. Com.*, superior or habenular commissure; *Sept. P.*, septum pellucidum; *S. N.*, substantia nigra; *Sp. Tr.*, spinothalamic tract; *Stri.*, striations of corpus striatum; *Sup. C.*, superior colliculus; *Sup. O.*, superior olive; *Th. d., thalamus,* dorsal surface; *Th. l.*, thalamus, lateral nucleus; *Th. M.*, thalamus, mesial nucleus; *Th. O. Tr.*, thalamo-olivary tract; *Th. O. Tr. l.*, thalamo-olivary tract forming fleece around inferior olive; *Trig. N. A.*, trigeminal nerve, afferent; *Trig. N. E.*, trigeminal nerve, efferent; *Troch. N.*, trochlear nerve; *T. S. C.*, taenia semicircularis; *Tub. C.*, tuber cinereum; *Ver.*, vermis; *V. Sp. Cbll.*, ventral spinocerebellar tract.

The Hemisphere Structures.—This hemisphere pocket presents at its base, resting on the lateral continuation (or paraterminal part) of the lamina terminalis: (1) the olfactory bulb and tubercle implanted anteriorly on the paraterminal substance, with the lateral olfactory root extending to the uncus and amygdaloid region, the posterolateral end of the hemisphere base; (2) the corpus striatum developed over the amygdala and paraterminal base; expanding over these structures, (3) the convex sheet of cerebral cortex, which extends between the lateral and the mesial olfactory gyri to form the convexity of the cerebral hemisphere across the lateral ventricle (fig. 2). The lateral ventricle with choroid plexus is comparable to the lateral pocket of the hindbrain which protrudes from the fourth ventricle, or ventricle of the hindbrain, between the flocculus and the tuberculum acusticum.

With this perspective of the development of the forebrain firmly established through the study of embryologic and comparative material, the student is prepared to begin the plasteline construction of the forebrain, using a definite color scheme for each system and indicating the functional and anatomic continuity of a mechanism by the use of the same color.

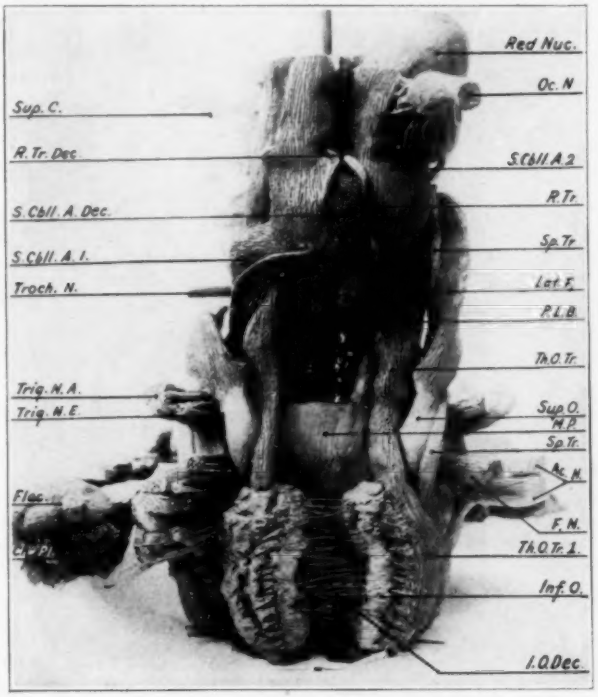


Fig. 3.—Ventral view. Reconstruction of part of the segmental and suprasegmental structures of the midbrain and hindbrain. It will aid in orientation to visualize this stage of the model with the cerebral peduncles, the pons bridge, the trapezoid body and the pyramids removed. The red nucleus on the right side is omitted; the right lateral fillet is cut off in order not to obscure the path of the rubrospinal tract; the flocculus with the choroid plexus is omitted on the left side. The upper limit of the model represents the cephalic limit of the midbrain; the lower limit of the model represents the caudal limit of the medulla.

PLASTELINE RECONSTRUCTION

The forebrain is built on the midbrain (figs. 4 and 5). Its construction is begun by laying down a frame for the midline structures as follows: the posterior commissure; the pineal recess and body; the superior or habenular commissure; the roof-membrane attached to the tenia of the epithalamus; the lamina terminalis extending between the anterior

commissure and the optic chiasm; the preoptic recess; the postoptic recess; the infundibulum; the tuber cinereum; the corpus mammillare, and the region of the interpeduncular ganglion. Thus provision is made for the roof, the anterior wall and the floor of the third ventricle (fig. 4).

The side wall is made up of the various parts of the thalamic mass: (1) the hypothalamus, (2) the thalamus proper or mesothalamus and (3) the epithalamus.

Hypothalamus.—A groove extending from the foramen of Monro to the pineal recess marks off the hypothalamus below from the meso-

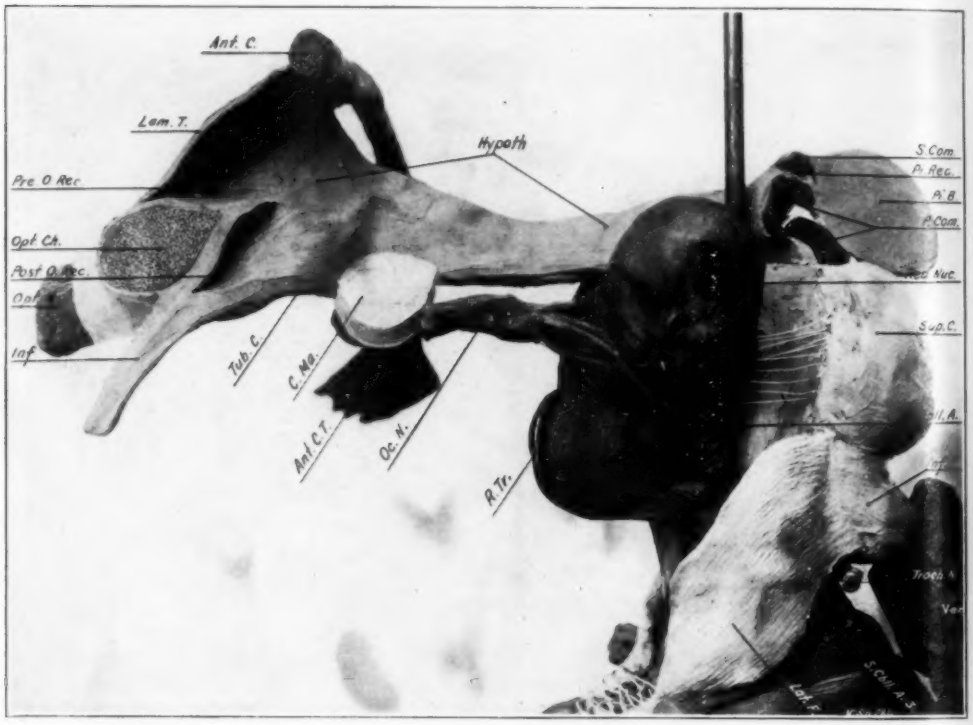


Fig. 4.—Lateral view of figure 3, from the left side, with the beginning of the reconstruction of the forebrain. The latter is built on the midbrain. In this and subsequent figures, the right side of the forebrain is shown; this figure represents a mesial view of the midline structures of the forebrain: anterior commissure, lamina terminalis, preoptic recess, optic chiasm, postoptic recess, infundibulum, tuber cinereum, mammillary body, hypothalamic plate, posterior commissure, pineal recess, pineal body, superior commissure; the roof membrane is not shown.

thalamus above. The former includes the following structures, mesially and laterally placed.

(a) Forming the Median Base (figs. 4 and 5): (1) The interpeduncular ganglion overlapping the cephalic end of the motor plate of the

midbrain; (2) the small spherical corpus mammillare on either side of the midline connected with the mesial olfactory gyrus complex by the fornix, with the anterior nucleus of the thalamus by the mammillothalamic bundle of Vicq d'Azyr and with the mastication segment through the mammillotegmental branches; (3) the tuber cinereum with the infundibulum containing various cell-groups apparently related to the metabolism of water and sugar in terms of which body growth may be consequently influenced.

(b) Forming the Lateral Base (fig. 6): (1) The lens-shaped corpus Luysii or hypothalamic nucleus with its capsule; (2) Forel's

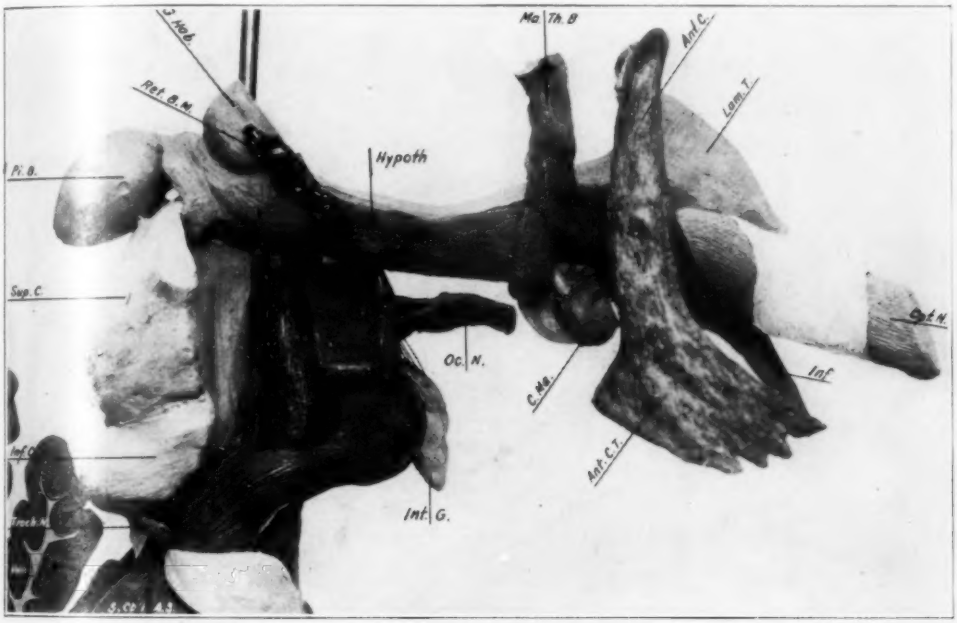


Fig. 5.—Lateral view of the structures of the forebrain represented in figure 4, with the addition of the ganglion habenulae, the retroflex bundle of Meynert, the interpeduncular ganglion and the mammillothalamic bundle of Vicq d'Azyr. The structures of the midbrain are viewed laterally from the right side; the red nucleus is omitted.

field *H* above it; (3) the substantia nigra of Soemmering below, sending a path to the tegmentum (these three structures together forming part of the striatal apparatus and all lying in the concavity formed by the crus cerebri), and (4) the red nucleus. The latter, while possibly a differentiation in the tegmentum of the midbrain is partly a station in the cerebellothalamic system and has an outlet in the rubrospinal (or more correctly the rubrosegmental) tract, which is influenced by both the cerebellar and the striatal organs. Topographically, the substantia

nigra and the red nucleus are often counted with the midbrain, but in many ways they are backward extensions of the hypothalamus and the mesothalamus respectively.

The Thalamus Body Proper or Mesothalamus (figs. 7, 8 and 9).—This forms the main thickness of the thalamic wall and is subdivided into the following parts:

1. The anterior nucleus bulges into the hemisphere ventricle just behind the foramen of Monro; it is possibly connected with the frontal lobe and with the striatum through the anterior part of the internal capsule and with the mammillary body through the bundle of Vicq d'Azyr.

2. The median nucleus, bordering on the third ventricle, consists of a mesial part lying beneath the taenia thalami and a posterolateral central

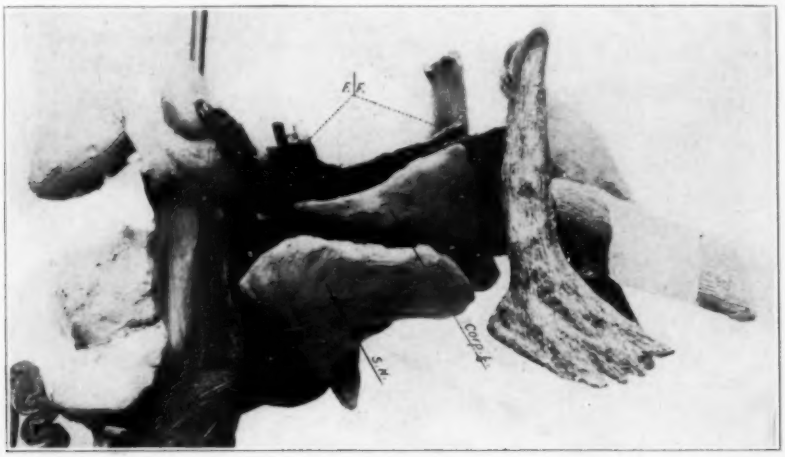


Fig. 6.—Same as figure 5, with the addition of the lateral base of the hypothalamus: Forel's field, corpus Luysii or hypothalamic nucleus, substantia nigra.

part, the centre médian of Luys, about the level of the ganglion habenulae. The median nucleus is connected with its fellow of the opposite side by the variable middle commissure (the massa intermedia) and with the frontal lobe by the anterior limb of the internal capsule; it is said (E. Sachs, 1909) also to be connected with the olfactory centers and with the caudate, but not with the cerebral cortex. The anterior and median nuclei, which phylogenetically constitute the older part of the thalamus, are separated by the internal medullary lamina from the lateral nucleus.

3. The lateral nucleus, of more recent origin, is divided into dorsal and ventral parts, which are further subdivided as follows: ventral *a* being the most posterior part; ventral *b* or the nucleus arcuatus forming

a shell around the base of the centre médian; ventral *c* constituting the end-station of the fillet and a more anterior ventrolateral group forming the terminal station of the rubral path. This lateroventral mass of cells is connected with the frontoparietal cortex by means of the knee and posterior limb of the internal capsule and receives from behind the fibers of the mesial fillet complex and of the superior cerebellar arm directly and indirectly through the red nucleus.

4. The posterior nuclei, or metathalamus (figs. 7, 8 and 9), also are of more recent origin, and consist of the internal and external geniculate

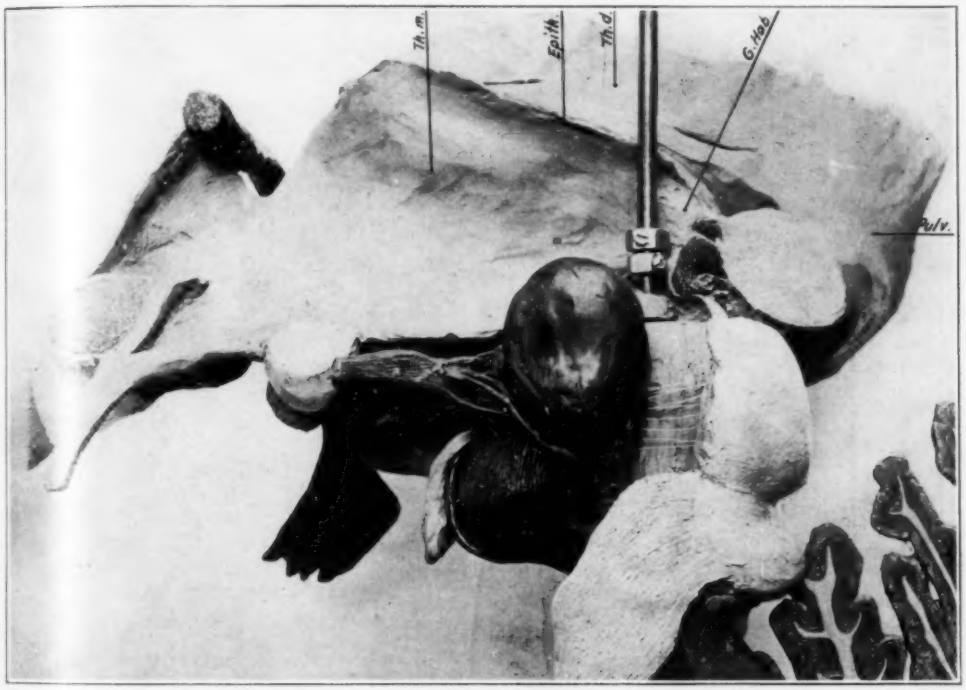


Fig. 7.—Mesial view of figure 6, with the addition of the structures of the lateral wall of the median forebrain (or third ventricle): the mesothalamus, the epithalamus, the metathalamus of pulvinar.

bodies overlapped by the pulvinar, the most posterior portion of the dorsolateral mass of the thalamus; the external geniculate body projecting at the lateral border of the pulvinar receives the optic tract (fig. 10) and the arm from the superior colliculus; it is connected with the visual area by means of the occipital radiation of the internal capsule and with the pulvinar by the geniculothalamic component; the internal geniculate body (overlapped by the pulvinar and partly covered by the optic fibers to the cappa cinerea of the midbrain) receives the arm of the inferior

colliculus (as brachium corporis quadrigemini posterioris or colliculi inferioris) and is connected with the auditory area by the sublenticular limb of the internal capsule.

The Epithalamus (fig. 7).—The epithalamus is made up of the ganglion and commissura habenulae receiving fibers (the taenia thalami or stria epithalami) from the nucleus basalis in the anterior perforated substance (the postolfactory or paraterminal area). It sends the retroflex bundle of Meynert, which sweeps ventrally and caudally along and through the mesial margin of the red nucleus, to the interpeduncular

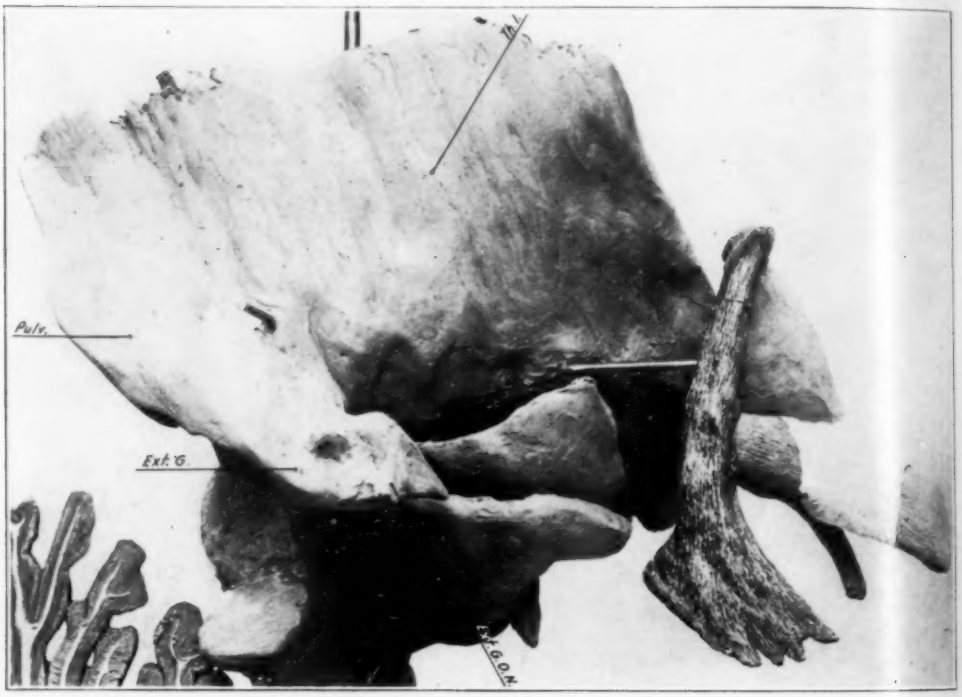


Fig. 8.—Lateral view of figure 7.

ganglion (fig. 5). From there, thin fibers run to the tegmentum of the mastication segment, a connection visible in the rat. The function of the epithalamic system in lower animals is probably concerned with reflexes of the olfactory snout, i.e., feeding reflexes.

With the thalamic mass in place, the third ventricle assumes the shape of a deep slit between the two thalami, communicating with the fourth ventricle through the ventricle of the midbrain or aqueduct and with the hemisphere or lateral ventricles through the foramina of Monro. Its roof is formed by an ependymal layer attached on either side to the taenia thalami of the epithalamic complex and posteriorly to the superior

commissure. In its posterior part the roof is drawn backward and upward so as to overlap the pineal body.

The choroid plexus, which is invaginated near the midline, passes anteriorly with the roof membrane to form the posterior wall of the foramen of Monro and thence continues into the hemisphere to pass over the dorsal surface of the thalamus; all along the line it connects the otherwise free fornix-fimbria of the hemisphere with the lamina affixa or the membranous floor which is attached to the dorsum of the thalamus along the stria cornea, the limiting line between thalamus and striatum. The choroid plexus membrane thus closes the lateral or hemisphere

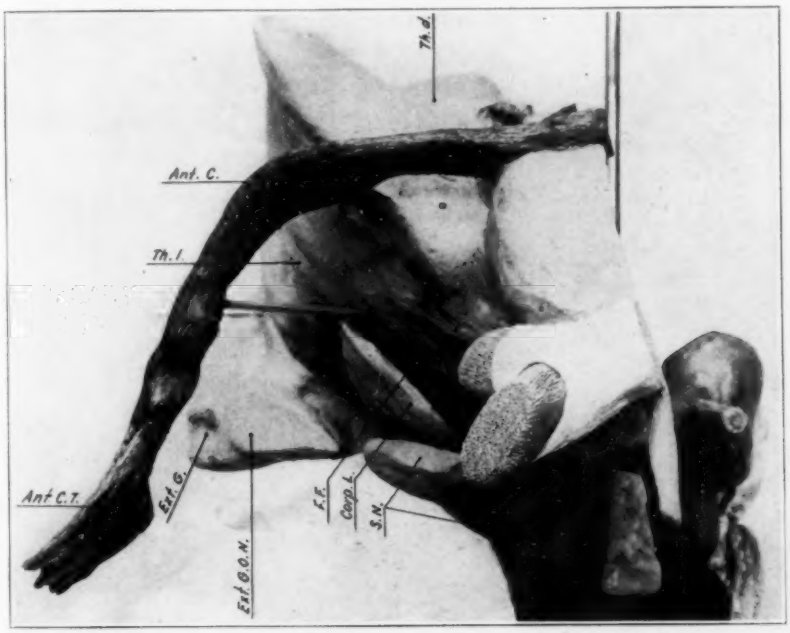


Fig. 9.—Frontal view of figure 8.

ventricle on its medioposterior side in a way which does not allow fibers from the mesial hemisphere edge to reach the base except through the fornix; the latter, as it passes forward from the whole length of the mesial olfactory gyrus, descends in front of the foramen of Monro (behind the anterior commissure) to enter the lateral side of the corpus mammillare with partial decussation.

The Optic Tract.—When the structures of the midline have been placed and the hypothalamus, the thalamus proper and the epithalamic complex constructed (figs. 4 to 10), the optic tract (fig. 10) is next put on extending from the optic chiasm to the external geniculate body,

loosely enough to let the crus cerebri or the downward continuation of the internal capsule pass to the basal surface when ready for this final addition.

The Olfactory Brain.—The next stage of the reconstruction embraces the formation of the olfactory brain, the corpus striatum and the cortex forming the convexity of the hemisphere. In relation to this phase, we review the development of the paraterminal body; in front of the optic chiasm and its tracts, the lamina terminalis spreads on either side into a layer called the anterior perforated substance or the postolfactory or

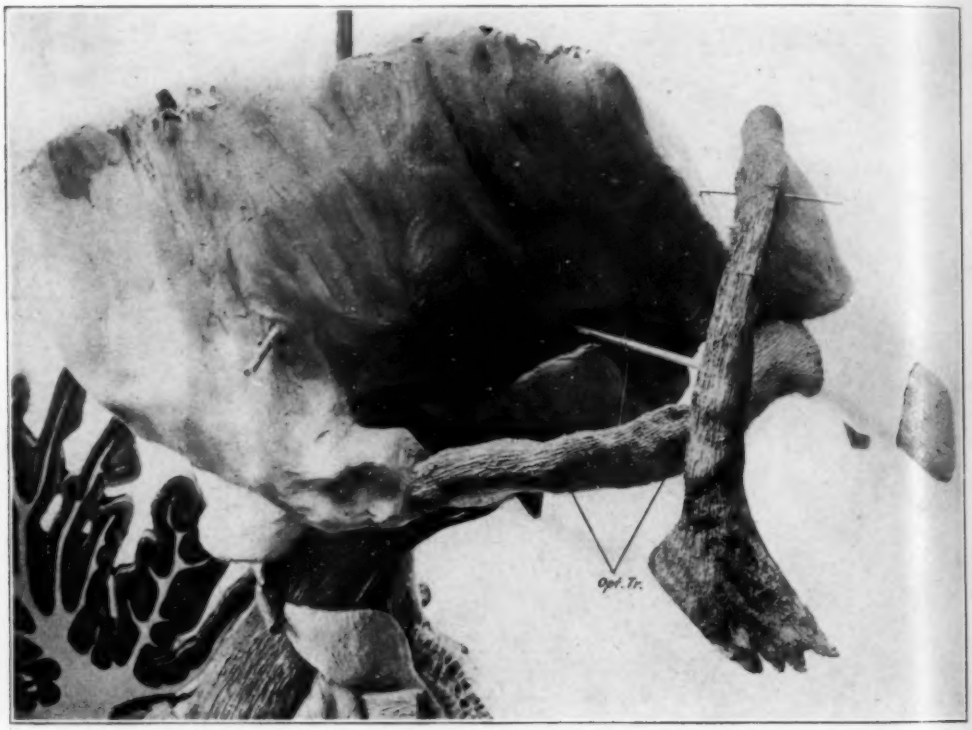


Fig. 10.—Same as figure 8, with the addition of the optic tract.

paraterminal area (with its pedunculus septi and nucleus basalis) and carries the interstriatal anterior commissure and the interhemispheric commissural mass, consisting of the corpus callosum, the hippocampal commissure, the septum pellucidum and the fornix column. This entire area, made up of the lamina terminalis and the substantia paraterminalis, the nucleus basalis, the massa commissuralis and fornix, constitutes the paraterminal body or postolfactory area. On this mass rests the olfactory protrusion consisting of the olfactory lobe, the olfactory tubercle, the lateral olfactory gyrus

extending to the amygdaloid nucleus in the uncus and the mesial olfactory gyrus, which is phylogenetically the oldest part of the cerebral cortex; the latter as it swings over the commissural mass to reach the posterior end of the amygdaloid region forms with the lateral olfactory gyrus the olfactory purse-string of the hemisphere, described by Gratiolet.

The construction at this stage is simplified by first putting down the amygdala as the lateral end of the base of the hemisphere, then the

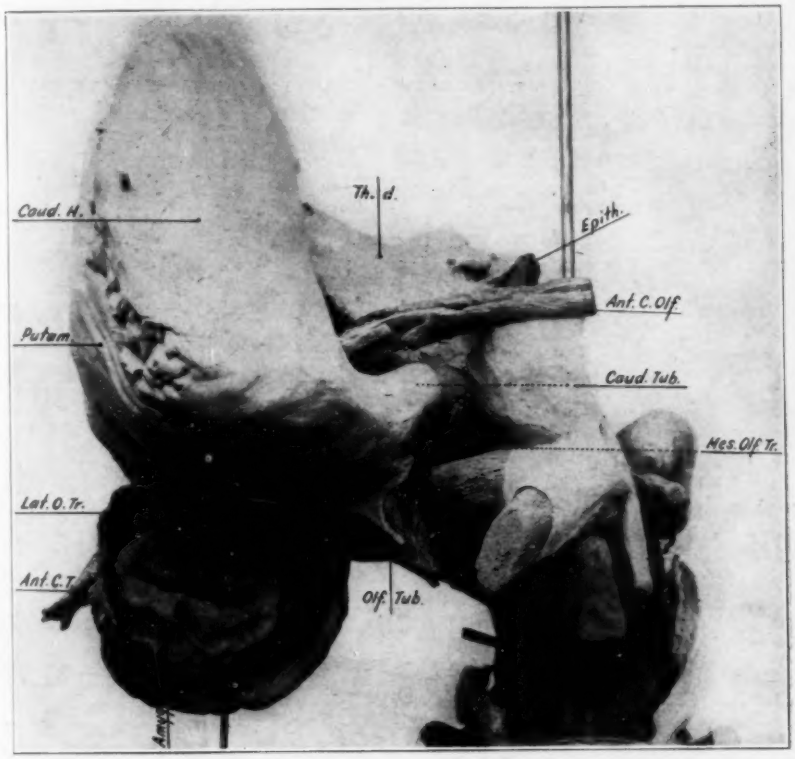


Fig. 11.—Same as figure 9, with the reconstruction of the hemisphere base added: the amygdaloid nucleus, the olfactory tubercle with the mesial and lateral olfactory roots and the corpus striatum; the olfactory portion of the anterior commissure has also been added.

corpus striatum developing over it and then the olfactory apparatus with its lobe, tubercle and lateral gyrus to the uncus or cortex-covering of the amygdala (figs. 11, 12 and 13). The mesial olfactory gyrus with the fornix and attached choroid plexus is put on later.

Corpus Striatum.—The corpus striatum consists of a large terminal cell mass partly divided into putamen and caudate nucleus by the internal

capsule, and a smaller and older wedge-shaped ventromedian body called the globus pallidus.

The globus pallidus, presenting mainly large cells, is the place of origin of the chief striatal efferent radiations: (*a*) the striato-luysian bundles from the globus to the corpus Luysii or hypothalamic nucleus, and (*b*) the ansa lenticularis to the thalamus, red nucleus, hypothalamic nuclei and substantia nigra. Many of the fibers end in the corpus Luysii, others pass dorsally and ventrally over its capsule to take part in the formation of the tegmentum or Haube, which Forel described and designated as field *H* (fig. 6).

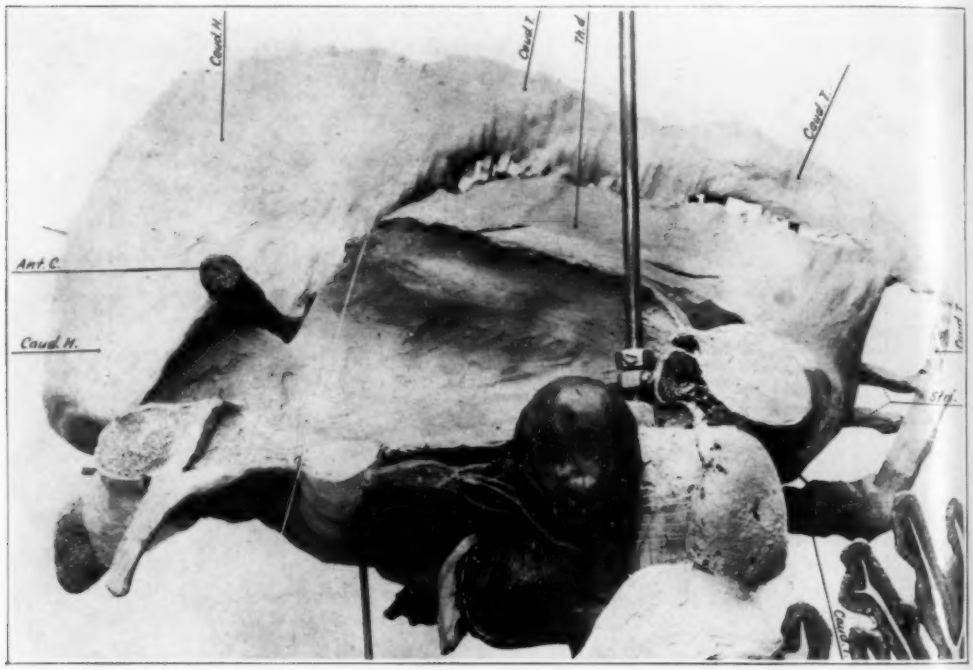


Fig. 12.—Mesial view of figure 11. Note the relation of the caudate head and tail to the anterior commissure and to the thalamus.

The term "lenticular nucleus" linking the globus pallidus with the putamen instead of combining the latter with the caudate is misleading, for the globus pallidus serves both these parts as an efferent system.

Paraterminal Body.—The next stage of the construction comprises the paraterminal body (figs. 14 and 15). It is begun with the placing of the fornix mass, which arises at the mesial edge of the cornu ammonis and sweeps mesially over the thalamus and around the foramen of Monro to terminate in the lateral part of the mammillary body (fig. 14). In its course it receives various designations; its fibers begin to arise

from the hook of the uncus and collect along the free border of the alveus of the cornu ammonis (the fibrous ventricular covering of the hippocampus) to form the fimbria; in its path over the dorsoposterior surface of the thalamus, where it is intimately applied to the splenium of the corpus callosum, it gives rise to the hippocampal commissure, while the fornix body and fimbria finally become condensed as fornix column behind the anterior commissure to form the anterior wall of the foramen of Monro.

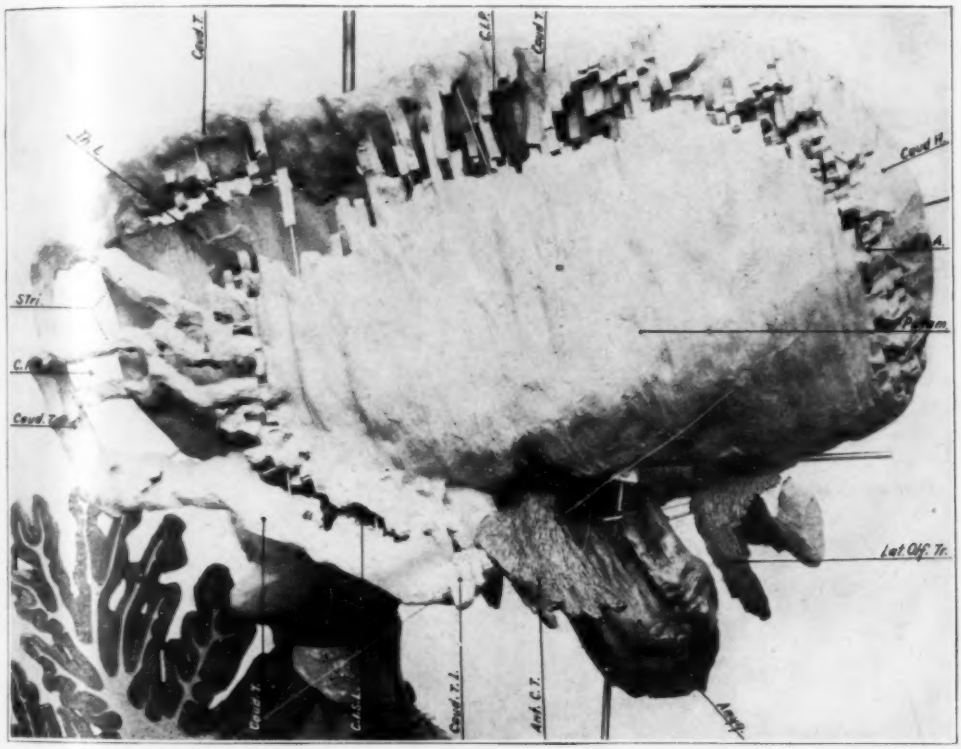


Fig. 13.—Lateral view of figure 12. The striations of the corpus striatum are plainly seen. Note the relationship of the tail of the caudate and the tail of the anterior commissure to the putamen and amygdaloid nucleus. Although part of the caudate tail may enter the amygdaloid nucleus, by far the greater portion terminates in the putamen. In this figure, the cleavage line of the internal capsule as it passes through the corpus striatum is also evident.

The "anterior perforated substance" is next added with the nucleus basalis and the pedunculus septi, the septum pellucidum and the commissural mass of the hemisphere with the corpus callosum (figs. 14 and 15). At this stage is included the mesial olfactory gyrus giving rise to the mesial wall radiation of the cortex, i.e., the fornix as already described.

The mesial olfactory gyrus itself extends as the tuberculocortical pathway between the olfactory tubercle and the uncus; in its course over the dorsal surface of the corpus callosum it is flattened and stretched into the striae Lancisii, the mesial stria swinging around the splenium to run to the uncus as dentate fascia, closely related to the palisade layer; at the uncus the dentate gyrus or fascia first enters the angle of the hook and then crosses the hood of the uncus as Giacommini's "bandelette de

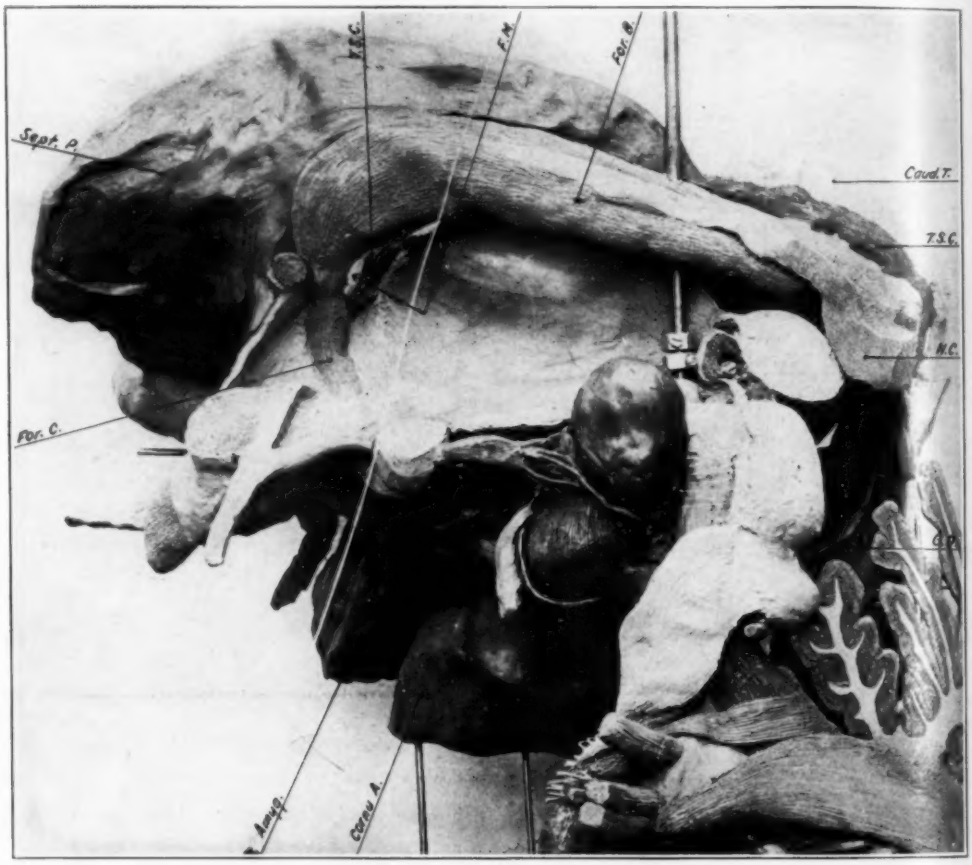


Fig. 14.—Same as figure 12, with the beginning of the reconstruction of the paraterminal body and the addition of the cornu ammonis. Note the hippocampal commissure, the fornix, the taenia semicircularis, the septum pellucidum, the cornu ammonis and the dentate gyrus. The reconstruction of the fornix brings into view the foramen of Monro.

l'hippocampe" to end on Meyer's "point of convergence." The olfactory bulb and tubercle with the complete mesial and lateral olfactory gyri, and the uncus as a connecting link between the two, has been likened by Gratiolet to a purse-string, with the cortex of the hemisphere as the

pouch. The mesial olfactory gyrus forms the posteromesial edge of the cortex, pushed down and partly overlapped by the neopallium which extends as the cortical convexity of the hemisphere between the mesial and lateral olfactory gyri.

The taenia semicircularis or stria cornea extends between the amygdala and the anterior perforated substance, marking the border between the thalamus and the caudate nucleus along the floor of the lateral ventricle from the foramen of Monro to the point of convergence.

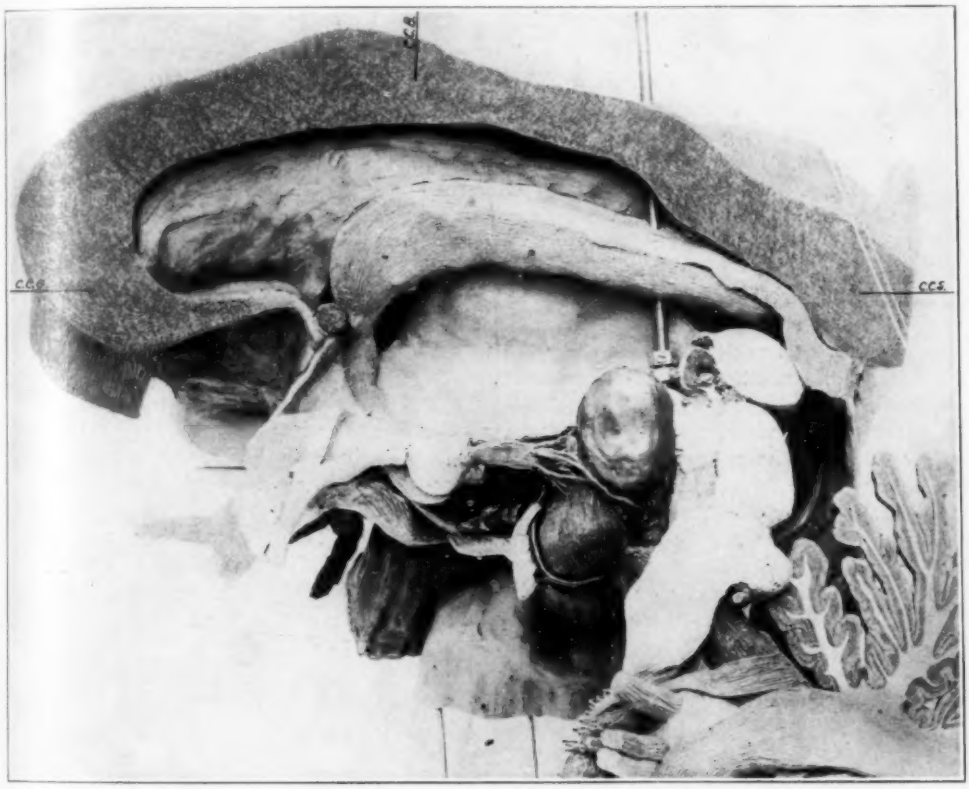


Fig. 15.—Same as figure 14, with the addition of the main commissural mass of the forebrain, the corpus callosum.

An easy review of the relations of the mesial olfactory gyrus, the corpus callosum with the septum, the fornix with the fimbria and the choroid membrane, and the stria cornea, is best obtained by starting from the point to which these structures converge, i.e., the uncus.

The uncus, lying just outside of and in front of about the middle of the optic tract, marks the posterior end of the hemisphere base, to which the following structures, which are readily found in a proper dissection

of the forebrain and easily traced in serial sections of the hemisphere, converge:

1. The caudate nucleus, which forms part of the floor and lateral wall of the lateral ventricle and is stretched backward over the dorsolateral margin of the thalamus, ending with a sharp bend near the point of convergence in the basal part of the putamen.

2. The stria cornea or taenia semicircularis with its vein, following the line of demarcation between the caudate nucleus and the thalamus, from the foramen of Monro to the uncus.

3. The lamina affixa and the choroid plexus, the latter extending from the roof of the third ventricle through the foramen of Monro along the fornix edge.

4. The fornix, as already described, carrying in the more anterior and dorsal part, the large commissural mass, which rests on the fornix and arises over the anterior commissure in a special spur from the lamina terminalis (above the neuropore level). By way of economy of distance, the fibers of the commissural mass arising from the neopallium pass across largely in the anterior, dorsal and posterior part of the commissural connection as corpus callosum. There are no fibers going across in the center of the massa commissuralis, which indeed becomes so stretched out longitudinally and laterally that it splits to form the cavity of the commissural mass, incorrectly called the fifth ventricle. In the course of development the two sagittal walls thus formed are approximated and form the septum lucidum, i. e., the mesial wall of the lateral ventricle, encircled by the corpus callosum anteriorly and dorsally, posteriorly and ventrally by the membrane extending between the two fornices from the splenium of the corpus callosum and the psalterium to the lamina terminalis, and thence anteriorly and ventrally from the lamina to the rostrum (fig. 15). The split or cavum between the two laminae of the septum is very plain in the fetus and even in the adult, particularly in the psalterium region (as "ventricle of Verga") and again more plainly in the hydrops occurring in some senile brains. In the osmatic animals no such split or cavum is produced. The septum lucidum in man, therefore, represents a previously distended and split part of the massa commissuralis in anosmatic animals. But the term is unfortunately also used for the paraterminal base of the mesial wall lying back of the mesial olfactory gyrus.

5. The mesial olfactory gyrus, consisting of the cornu ammonis and stria Lancisii, beginning in the olfactory tubercle and ending in the cortex of the uncus.

Cerebral Cortex.—With this foundation of a general knowledge of the olfactory apparatus, the corpus striatum and the commissural mass, a review of the cerebral cortex can be presented. Extending between the mesial and lateral olfactory gyri is the convexity of the hemisphere—the neopallium. The sessile portion or island of Reil, the frontal lobe (in most mammals overlapping and outgrowing the olfactory bulb) and the parieto-occipitotemporal expanse, existing as a folded sheet with areas of more or less clearly differentiated lamination, mark anatomically distinct regions. The neopallium gives rise to the radiation of the lateral wall, i.e., the internal capsule, which pierces the caudate-putamen end-mass to pass along the lateral wall of the thalamus to the basal surface as

the crus cerebri, emerging beneath the optic tract. Functionally and structurally distinct are found: (1) The mesial olfactory gyrus and uncus related to the olfactory and gustatory (?) functions. (2) The calcarine cortex as the receiving center of the optic radiation, which consists of the internal and external sagittal layers; the stratum sagittale externum passes from the external geniculate to the occipital area, the most ventral fibers forming a detour around the tapetum in the temporal lobe, whereas the stratum sagittale internum consists of efferent fibers from the occipital cortex to the pulvinar and tectum mesencephalicum and through the lateral part of the crus to the pons, thence influencing the cerebellum.[3] (3) The auditory receiving station in the transverse temporal gyrus, connected with the internal or mesial geniculate body by the sublenticular auditory radiation. (4) The kinesthetic area consisting of the postcentral parietal cortex for general sensibility, and the anterior central gyrus for the forebrain efferent or corticosegmental paths—i.e., the pyramidal and corticobulbar tracts. Many of these areas of the cortex and the intermediate portions have definite connections with definite cell groups of the thalamus.

In addition to these "projection" centers, there may be distinguished: (1) the posterior association field, consisting of the bulk of the temporal and parietal cortex; (2) the island of Reil, and (3) the frontal association field.

The fibers connected with this whole expanse of cortex are arranged as: (1) white matter within the gyrus; (2) diffuse subcortical white matter or centrum ovale, with short and long association bundles; (3) from the stratified deep layers, the fibers of: (*a*) the corpus callosum (the dorsal interhemispheric commissure), and (*b*) the corona radiata and capsula interna, constituting the lateral wall radiation of the hemisphere.

The capsula interna presents the following coarse morphologic subdivisions (fig. 13): (1) the anterior limb (*cia*) containing the frontal radiations; (2) the knee, also made up of frontal radiations and not, as is usually assumed, a landmark for the central efferent facial path, which lies further back; (3) the posterior limb (*cip*), containing partly frontal radiations but mainly the radiations of the kinesthetic area, marked off laterally by the most posterolateral fibers of the striatohypothalamic bundles crossing to the corpus Luysii; (4) the retrolenticular part (*cirl*), with the parieto-occipital radiation; (5) the sublenticular part (*cisl*), for the temporal connections.

The pes pedunculi forms a simple shell, containing in its shallow convexity the substantia nigra and corpus Luysii. In connection with

3. For the details for these connections see Meyer, Adolf: The Transactions of American Physicians, 1907.

the latter, fibers of the ansa lenticularis pass as striatoluysian bundles around and through the internal capsule from the globus pallidus to the corpus Luysii and its dorsal capsule and the field *H* of Forel. The most lateral part of the cerebral peduncle is formed by the occipitotemporopontile bundle (of Türck).

Returning now to the reconstruction: Having placed the corpus striatum, the paraterminal body and the mesial olfactory gyrus and radiation, one next adds the cerebral afferent tracts consisting of the auditory and visual paths and the sensory fillets (mesial and superior) to the thalamus; from here the thalamocortical system and finally the efferent tracts consisting of the corticopontile and corticosegmental systems. The corticobulbar system joins the mesial fillet.

Thus, in the forebrain one recognizes essentially two terminal structures of the neural tube—the striatum and the cerebral cortex—they are end-stations. The thalamus is treated as a relay station with some terminal functions of its own.

SUMMARY

In connection with the plasteline construction, a complete series of Weigert-stained transverse, horizontal and sagittal sections is used, and the student is instructed to follow up the parts placed on his model as definite mechanisms or systems, making summary recapitulations as follows:

The Median Line Structures.—The posterior commissure, the pineal recess and pineal body, the superior commissure, the taenia and roof membrane with choroid plexus, the region of the neuropore, the anterior commissure, the lamina terminalis, the optic recess and chiasm, the infundibulum and pituitary body, the tuber cinereum, the mammillary region and the interpeduncular ganglion.

The Lateral Walls of the Median Forebrain or Third Ventricle.—These consist of the thalami and the olfactory and hemisphere protrusions, carrying also the corpus striatum and the paraterminal body.

1. The Thalamic Mass: The thalamic masses lie between the posterior limb of the internal capsule and the ventricle of the median forebrain, and present three subdivisions: (*a*) The epithalamus consisting of the stria epithalami, ganglion habenulae, the superior commissure and the habenulo-interpeduncular bundle to the interpeduncular ganglion, which in turn is connected with the tegmentum of the anterior hindbrain segment. (*b*) The thalamus proper, or mesothalamus, consisting of the anterior, the median, the lateroventral and the posterior nuclei with the pulvinar; the ventral region being continued into the region of the midbrain as the red nucleus. (*c*) The hypothalamus, consisting of the corpus Luysii and the substantia nigra (as striatal dependent parts) and the

corpus mammillare (dependent on the mesial olfactory gyrus) and the tuber cinereum with the infundibulum and the postoptic recess forming the median recess.

2. The Corpus Striatum: A terminal body consisting of the putamen-caudate mass (partly split by the internal capsule) and the globus pallidus, with the striatoluysian and ansa lenticularis connections for the corpus Luysii, substantia nigra, Forel's field and the red nucleus.

3. The Olfactory Mechanism: The olfactory lobe, the olfactory tubercle, the lateral olfactory root (ending in the cortex of the amygdala) and the mesial olfactory gyrus; the latter extending as the dorsomesial border of the hemisphere pouch around the dorsal surface of the corpus callosum to the cornu ammonis, which gives origin to the fornix, the mesial wall radiation of the hemisphere.

4. The Paraterminal Body: The anterior perforated substance with the nucleus basalis and the pedunculus septi, the commissural mass made up of the corpus callosum with the septum pellucidum as its cleavage product, and the hippocampal commissure and decussation as part of the fornix mass, extending from the cornu ammonis to the mammillary body.

5. The Cortex of the Neopallium: Included with this are the corona radiata or lateral wall radiation and its connections with the thalamus and the peduncle.

In the drawings that the student is required to make of his histologic section studies, a definite color scheme, which is also applied to the plasteline reconstruction, is used to represent systems that have a functional and anatomic continuity:

Olfactory	segmental—blue suprasegmental—yellow
Paraterminal body	dark red
Optic	segmental—blue suprasegmental—green
Auditory	segmental—blue suprasegmental—violet
Corpus striatum	Caudate and putamen—dark blue Globus pallidus—light red
Forebrain efferent	carmine
Forebrain afferent	green
Cerebellar mechanisms	afferent—orange efferent—brown nuclei and red nucleus—brown secondary efferent systems—black
Segmental	efferent—brick red afferent—blue

ABSTRACT OF DISCUSSION

Dr. Adolf Meyer: With regard to this problem it is probably difficult to understand from a mere review just what it means from the point of view of instruction of the student. The instruction given in this course calls on the

student for a simple practical demonstration of what he has acquired. It is based on principles of simplification without schematization; they are of tremendous importance, as far as I can see, particularly in these days when the courses are being reduced to minimum presentations.

Dr. Hausman has the great advantage of having the whole first-year class at Cornell undisturbed by preliminary training under the influence of other concepts. I have the other condition, namely, I get my first-year students for elective work after they have had what might be called a full lecture and section course, after they have accumulated a tremendous vocabulary and while they are experiencing great difficulty in getting a unified conception of the material that has been brought before them. The conception of the course is based on taking the things which we see every day in our examination of patients: the olfactory segment, the eye and its mechanisms; the isthmus and the zygoma indicating the separation of segments of the midbrain and hindbrain; the latter are the mastication segment, the segment of the ear and its annexes, including the abducens that pulls the eye in the direction of the sound; then the segments of the viscera of the mouth and neck, etc., and then the segments of the neck and trunk. That is put before the students in the form of a problem: How do you divide the head? What neural mechanisms is the head integrated by? After they have acquired an actual conception of the subdivisions of the head which they are constantly meeting and using and of the early stages of the fetal brain in His's models, they are led to make a connection between those relatively simple things, and after that has been thoroughly digested and the motor and receptor plates have been constructed in three dimensions, one can go on to build the cerebellar apparatus around the segmental foundations, next the suprasegmental apparatus of the midbrain, and finally the apparatus of the forebrain, with constant reference to the parts of the body and their uses and the fetal and adult brain and the sections. Mere deductions from terminology are discouraged. It is always a physiologic need that is emphasized, definite parts of the body and their functions and their neural integration. There is little terminology: First, specific functional necessities, and then the determination of the pertinent structures in actual specimens; discussion from an embrylogic, morphologic, experimental and pathologic anatomic-functional point of view, and with that the building up of the parts in plasteline. Any student ought to be able to recapitulate in twenty or thirty minutes the essential points of the nervous system so represented, so as to show an instructor whether he has or has not got the foundations of the nervous system.

Dr. Hugh T. Patrick, Chicago: This is one of the most beautiful and logical demonstrations I have ever seen. I understand all first-year students take this course. How long a time do they spend on it?

Dr. Louis Hausman: The students spend about eighty hours on neurologic anatomy and histology; of the eighty hours about thirty-five are given to the reconstruction. The reconstruction course is greatly simplified for them and the actual mechanical work is made easy. The rest of the time is given to actual gross dissections of the brain, the study of microscopic serial sections, and then the drawing of those parts that have been put on the model itself.

Dr. Patrick: I would not wish to put myself in the position of passing finally on this, but if first year medical students are required to learn all of these anatomic details of the brain, it strikes me at this minute that this is a pedagogic crime.

DR. MEYER: I take no offense at this perfectly outspoken comment because it comes from a friend who evidently does not know as yet how the course is managed; but I want to say for the relief of those who might suspect a "pedagogic crime" that in my Hopkins course thirty-two hours are given to this matter; those hours are spent with a great deal of mutual discussion and comparison of the results, and, as far as I am aware, there have not been any great disasters; after all, the student is urged to help himself to the material and facts he actually feels a need for. As long as the student is forced to talk and to show what he is talking about, after having seen and studied and handled things, I do not believe that we have much of a pedagogic crime.

THE VENTRICULAR SYSTEM

ITS RELATION TO THE CEREBELLUM: VENTRICULOGRAPHY AND OTHER VENTRICULAR EVIDENCE IN THE RECOGNITION OF CEREBELLAR DISEASE *

CHARLES A. ELSBERG, M.D.
AND
SAMUEL SILBERT, M.D.
NEW YORK

Studies of the dilated ventricles, made visible on the roentgenogram by the injection of air (ventriculography, pneumography, encephalography), have contributed greatly to the understanding of the changes in the size and shape of the ventricular cavities which result from obstructing lesions in the posterior cranial fossa. The estimation of the size of the ventricles from the amount of fluid they contain, the information gained—by the intraventricular injection of colored dyes—of the patency of the pathways of the ventricular and subarachnoid channels, and finally, studies of models of the basal cisterns and of the ventricles have greatly furthered this knowledge.

That occlusion of the foramina of Magendie [1] and Luschka, of the fourth ventricle, and of the aqueduct of Sylvius is followed by enlargement of the lateral and third ventricles from the retention of cerebrospinal fluid has been known for many years. During the last few decades, studies have made it possible to differentiate this ventricular dilatation from the distention that occurs in expanding lesions which cause pressure on the third ventricle or on some part of one or both lateral ventricles.

Enlargement of both lateral ventricles may be caused by supratentorial as well as infratentorial tumors. New growths which involve the central ganglia may cause a marked bilateral ventricular dilatation from pressure on the third ventricle, but in these growths there is usually more or less distortion and dislocation of some part of the ventricular system. Tumors in the third ventricle or on its floor—pineal tumors, tumors of the craniopharyngeal pouch and large pituitary growths—may compress the ventricle and cause an obstructive hydrocephalus of both lateral ventricles. Compression of the foramina of Monro by new growths above, behind, below or in front of these openings may bring about the same result.

---

* Read at the Annual Meeting of the Association for Research in Nervous and Mental Disease, December, 1926.

1. We shall not enter into the question of whether the foramen of Magendie is, in actual fact, an opening.

Dilatation of both lateral and third ventricles, and even of the iter and fourth ventricle, is frequent in other disease in the posterior cranial fossa—in closure of the foramina of Magendie and Luschka as the result of basilar meningitis; in inflammatory changes in the subtentorial leptomeninges in the course of encephalitis; in adhesive processes in the basal cisterns (cisternal arachnoiditis of Cushing and Horrax); in hydrocephalus secondary to spina bifida; in some instances of senile atrophy of the brain, and in those poorly understood and, up to the present, therapeutically hopeless varieties of congenital hydrocephalus in which demonstrable evidence of obstruction in the intracranial and spinal pathways of the cerebrospinal fluid is not present.[2]

Dilatation of the lateral and third ventricles due to tumors in the posterior cranial fossa occurs, in the majority of instances, as soon as the growth has begun to cause pressure on the fourth ventricle or the aqueduct. In some instances, however, in spite of the pressure of a large intracerebellar or extracerebellar tumor, the lateral ventricles are not appreciably dilated. In several recent instances of tumor of the pons, which came to autopsy, we were surprised to find that the ventricles were not dilated (fig. 1).

Therefore, it is well to remember that the absence of ventricular dilatation, although infrequent, does not actually negate the possibility of the presence of an expanding lesion of considerable size in or about the cerebellum, pons or medulla. In patients with cerebellar disturbances, in whom it is uncertain whether the disease is of an expanding nature, the surgeon often has to puncture the lateral ventricle in order to determine whether or not the ventricles are dilated. If the lateral ventricles contain a greatly increased amount of fluid, the presence of a new growth is more likely. If, on the other hand, only a small amount of fluid—from 5 to 10 cc.—is obtained by ventricular puncture, it is probable but not certain that a subtentorial expanding lesion is not present but that the condition is caused by an intrinsic vascular or degenerative cerebellar disease.

MECHANICAL FACTORS IN VENTRICULAR DILATATION RESULTING FROM CEREBELLAR NEW GROWTHS

Dilatation of the lateral ventricles may be caused by intraventricular tumors (fig. 2) which either partly or completely interfere with the circulation of the cerebrospinal fluid or which are similar in action to that of a ball valve. In the latter instance, the repeated periods of

2. There is little proof that in these cases of so-called hydrocephalus due to hypersecretion the epiphysis is often atrophied. The interesting suggestion was made by O. Foerster that atrophy of the pineal body may occur, and that a function of the pineal body is to inhibit the secretory activity of the choroid plexuses through its effect on the hypophysis.

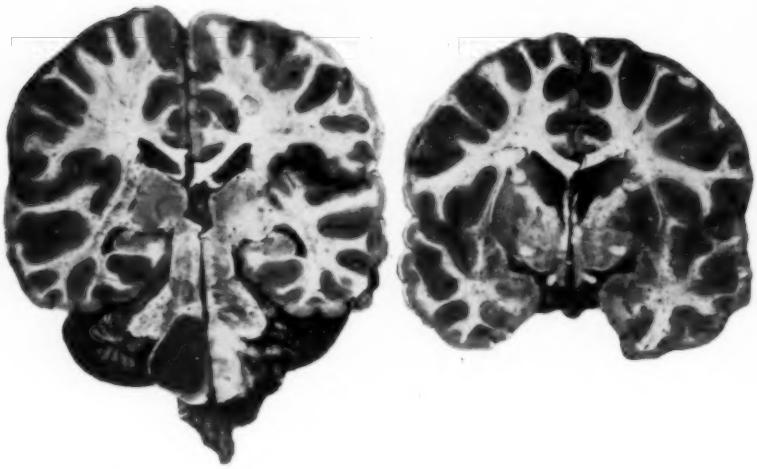


Fig. 1.—Tumor of the pons with little ventricular dilatation.

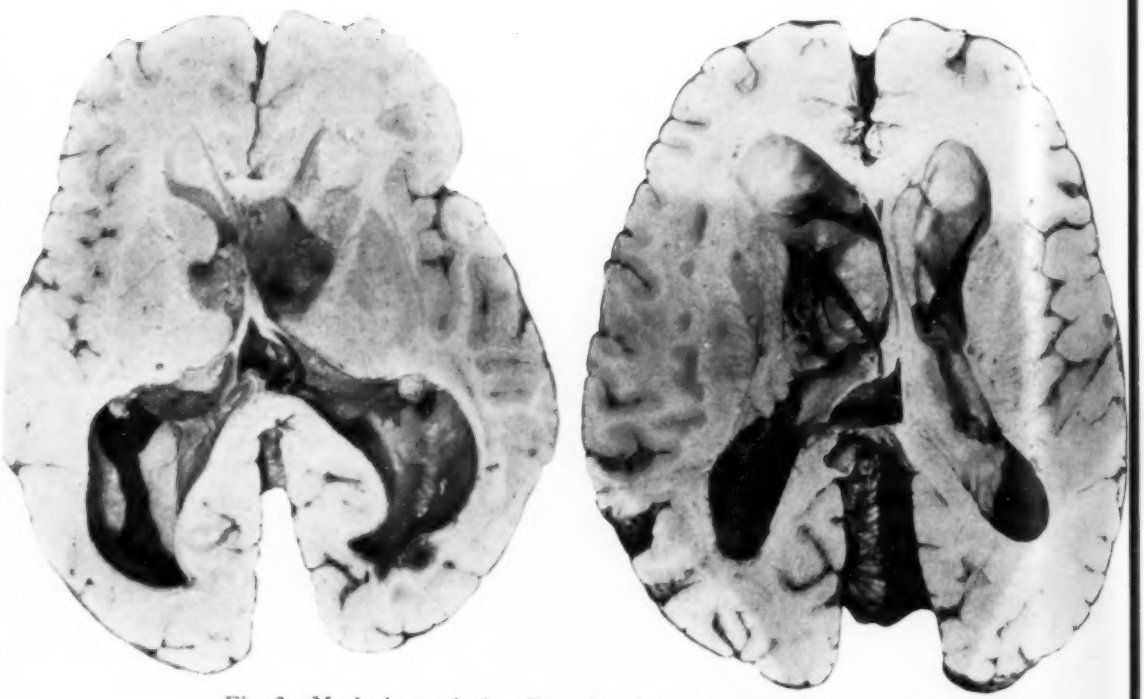


Fig. 2.—Marked ventricular distention from intraventricular tumors.

obstruction may finally be followed by a degree of hydrocephalus as great as is often observed in complete obstruction of the iter or fourth ventricle.

Bilateral dilatation of the ventricles will often follow even when the pathways are only partially blocked by a neoplasm of the posterior fossa. However, there is another factor to which too little attention has hitherto been paid. The dilated ventricles themselves may press on or distort the foramina of Monro, the third ventricle and perhaps the aqueduct, and may thus contribute to the increase of the hydrocephalus. A dye which has been injected into a lateral ventricle and which did not pass the obstruction and appear in the lumbar puncture fluid will sometimes promptly pass down into the spinal subarachnoid space after considerable fluid has been removed from the ventricles. There may be therefore a sort of vicious circle in increasing ventricular distention.

After the removal of considerable fluid (from 50 to 100 cc. or more) from a dilated ventricle, the relations of a tumor to the aqueduct of Sylvius or to the fourth ventricle may become altered: either the growth may cause more pressure on some part of these pathways or the compression may be diminished. It is not mere speculation to believe that a tumor in the substance of the vermis or cerebellar lobe or in the pons may make greater pressure on the fourth ventricle after cerebrospinal fluid has been removed, and it is well known that sudden death may follow the removal of fluid from the lateral ventricles in growths in these locations. On the other hand, tumors in the posterior cranial fossa that are firmly fixed to the bone or dura (growths in the cerebellopontile angle, tentorial endothelioma, etc.) may for a time make less pressure on the cerebellum and the ventricular pathways after the distended lateral ventricles have been more or less completely emptied of fluid.

DANGERS OF LUMBAR PUNCTURE IN SUBTENTORIAL EXPANDING LEGIONS

That there is an element of danger in the removal of cerebrospinal fluid by ventricular puncture in new growths of the pons or vermis is well known. For many years, the greater danger from the removal of fluid by lumbar puncture in expanding disease of the posterior fossa has been recognized, and therefore lumbar puncture is contraindicated if the presence of a cerebellar tumor is suspected. The withdrawal of even a small amount of fluid from the lumbar sac removes a certain amount of support of the structures of the posterior fossa and is apt to be followed by the herniation of the cerebellar lobes through the foramen magnum, with consequent pressure on the medulla. The pressure cone, which is visible in the cerebellar lobes in such cases, is well known. It is questionable, however, if this danger is great when considerable fluid has first been removed from the ventricles by ventricular puncture before

the lumbar puncture is performed, and it is interesting that we have never observed medullary symptoms after lumbar puncture and removal of fluid in the noncommunicating form of hydrocephalus not due to tumor of infancy and childhood. In these patients we have frequently removed considerable cerebrospinal fluid by lumbar puncture without the production of any harmful symptoms.[3] The presence of a tumor in the posterior cranial fossa is probably the important factor in foraminal herniation of the cerebellar lobes with compression of the medulla and sudden respiratory failure.

It is fairly probable but is not yet definitely proved that the danger of a lumbar puncture in cerebellar tumors is small if the fluid removed by lumbar puncture is at once replaced by air.

ALTERATIONS IN SIZE AND SHAPE OF LATERAL VENTRICLES IN SUBTENTORIAL DISEASE AS SEEN ON VENTRICULOGRAM

The general size and shape of the dilated ventricle in lesions of the posterior fossa are so well known, both from the pictures obtained by the injections of air and the roentgen ray and from postmortem studies, that little time need be devoted to this part of the subject. As seen in the ventriculograms and in models of the ventricles (figs. 3 and 4), the dilated ventricles are regular in outline. In unilateral cerebellar tumors, the lateral ventricle on the side opposite that of the tumor is distinctly larger than that of the same side (figs. 5 and 6), unless the dilatation is extreme. Because of this difference between the size of the two lateral ventricles, it is often possible to determine the side on which the growth occurs from the study of the roentgenograms.

The best way to measure with exactness the difference in cubic content between the two lateral ventricles would be to make models of the ventricles and then to determine their actual and comparative size by the displacement of water method. We have done this in a few instances, but we were unable to make a sufficient number of such observations because of our unwillingness to destroy the wax models.

We have, however, made careful measurements on the ventriculograms of the average width of various parts of the ventricles. It is hardly necessary to state that in lateral views of the ventricles the measurements of the right lateral ventricle were only made on films taken from the right, and of the left ventricle on the films that had been taken from the left. While there is a margin of error in the actual figures obtained on account of the exaggeration of the size of the ventricles because of the distance between the roentgen-ray tube and the

3. In obstructive hydrocephalus of infancy due to closure of the iter, considerable cerebrospinal fluid may be present in the basal cisterns. This is the explanation of the fact that, at times, in infants, as much as 30 to 50 cc. or more of fluid can be withdrawn by lumbar puncture.

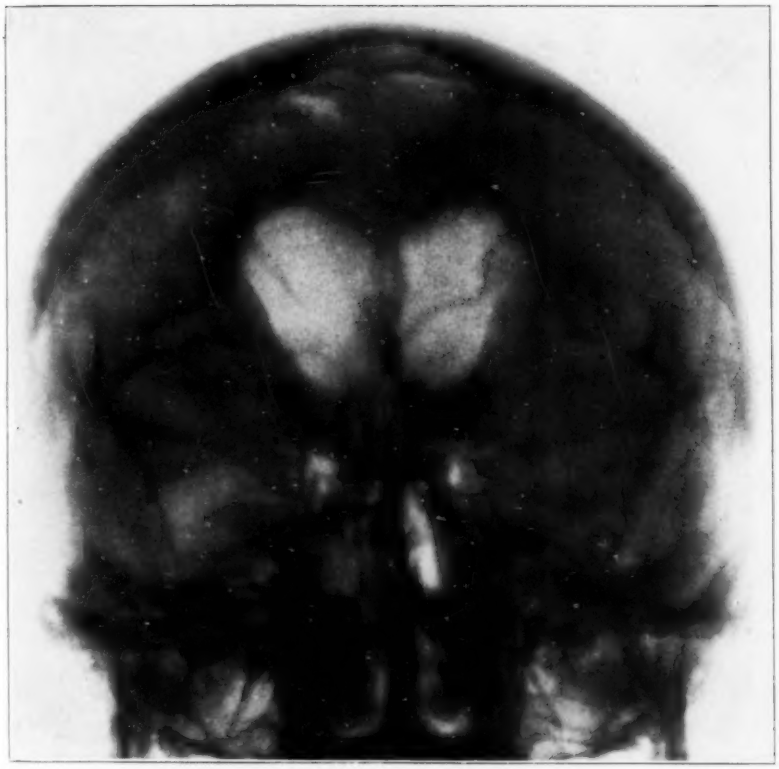


Fig. 3.—Ventriculogram of a patient with dilated lateral ventricles showing that the ventricles are regular in outline.

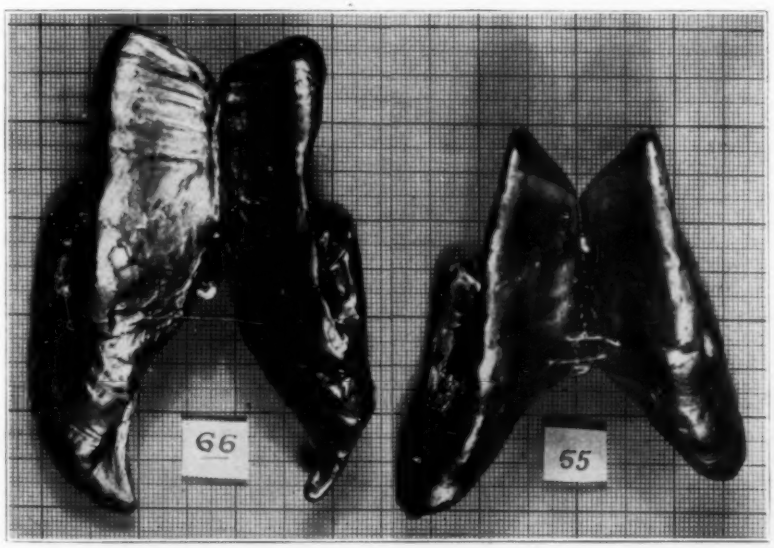


Fig. 4.—Wax models of the dilated ventricles in two cases of tumor of the lateral recess showing the regular outline of the ventricles.

Fig. 5.—Anteroposterior ventriculogram showing greater enlargement of contralateral ventricle in a case of right cerebellar tumor.

Fig. 6.—Anteroposterior ventriculogram showing greater enlargement of the contralateral ventricle in a patient with left cerebellar tumor.

head, this error is constant, and when comparative measurements are made, is insignificant. The figures that we obtained are given in the accompanying table.

In the early stages of obstructive hydrocephalus caused by tumor or inflammatory disease in or about the cerebellum, all parts of the lateral ventricles—the body and the frontal, temporal and occipital horns—enlarge more or less equally. When the dilatation has become marked, the inferior horns will be found to be relatively small as compared with the remainder of the ventricles (figs. 7 and 8). Our first studies led us to suspect that this difference in size between the bodies and frontal horns as compared with the temporal horns was always characteristic of infratentorial as opposed to supratentorial disease. The study of a larger series of cases has, however, shown that this difference in size is distinct only in patients with advanced dilatation of the lateral ventricular system

Measurements of the Comparative Size of the Two Lateral Ventricles in Five Instances of Unilateral Cerebellar Tumor

Part of Ventricle	Average Width in Centimeters of		Average Greater Size in Centimeters of	
	Homolateral Ventricle	Contralateral Ventricle	Homolateral Ventricle	Contralateral Ventricle
Frontal horn	3.25	3.75	0	0.5
Occipital Horn	3.5	4.1	0	0.6
Temporal horn	1.7	1.9	0	0.2
Atrium	5.3	6.1	0	0.8

which is so often associated with cerebellar tumors, and that it therefore has diagnostic significance only in advanced subtentorial as compared with supratentorial disease.

On the ventriculograms, the dilated lateral ventricles due to subtentorial expanding lesions are regular in outline. Any abnormality in their form should make one suspect that the lesion is supratentorial even though there is evidence that the obstruction is at the aqueduct of Sylvius. In a patient recently studied at the Neurological Institute, who had symptoms and signs that pointed to a tumor of the posterior fossa, the injection of air into one lateral ventricle showed that both lateral ventricles were greatly dilated and that the foramina of Monro were patent because air passed freely from the one into the other ventricular cavity. The marked dilatation of the lateral and of the anterior part of the third ventricles seen on the lateral ventriculograms was like that usually seen in tumors of the posterior fossa, but the defects in the outlines of the ventricles seen on the anteroposterior roentgenograms (figs. 9 and 10) demonstrated that the new growth lay on the base of the brain above the tentorium. The postmortem examination showed that the patient had a large tumor on the base; it extended from the optic chiasm in front to the pons behind (figs. 11 and 12), and had

Fig. 7.—Ventriculogram of a patient with a cerebellar tumor and advanced ventricular distention, showing the differences in size between the frontal horn and body, and the temporal horn.

Fig. 8.—The relative size of the anterior horn and body to the inferior horn in a case of obstructive hydrocephalus from a lesion of the posterior fossa.

caused an obstructive hydrocephalus by pressure on the hind part of the third ventricle and the aqueduct of Sylvius.

In unilateral cerebellar tumors or tumors of the cerebellopontile angle, the homolateral occipital horn is often but not always dislocated forward, upward and outward, and this difference between the position

Fig. 9.—Anterior ventriculogram showing defect in the lower border of the lateral ventricles from a tumor on the base of the brain. This is the same patient that is shown in figures 11 and 12.

of the posterior horns of the ventricles can be recognized and the side on which the lesion of the posterior fossa is present suspected from the careful study of the ventriculograms (figs. 13, 14 and 15). This dislocation of the homolateral posterior horn has been noted on the operating table by others and by us. Unless the lateral ventricles are greatly

distended, the occipital horn on the same side as the tumor can best be reached by pointing the needle more outward and upward than would usually be done in puncture of the posterior horn of a lateral ventricle from the occipital region.

Fig. 10.—Posterior ventriculogram of patient in figure 9.

When it is present, this change in the position of the homolateral posterior horn is of significance in support of clinical signs which point to the cerebellar lobe of the same side, but the side on which the tumor exists should only be suspected from the ventriculogram if no other facts are present to support a diagnosis of the side of the lesion.

In a recently published report of studies of the ventricles, we have called attention to the fact that in many infiltrating growths of the cerebral hemispheres the vertical planes of the bodies and of the frontal and temporal horns on the side of the tumor are further apart, while the corresponding horns on the opposite side are closer together than normal. This change in relations of these parts of the lateral ventricles

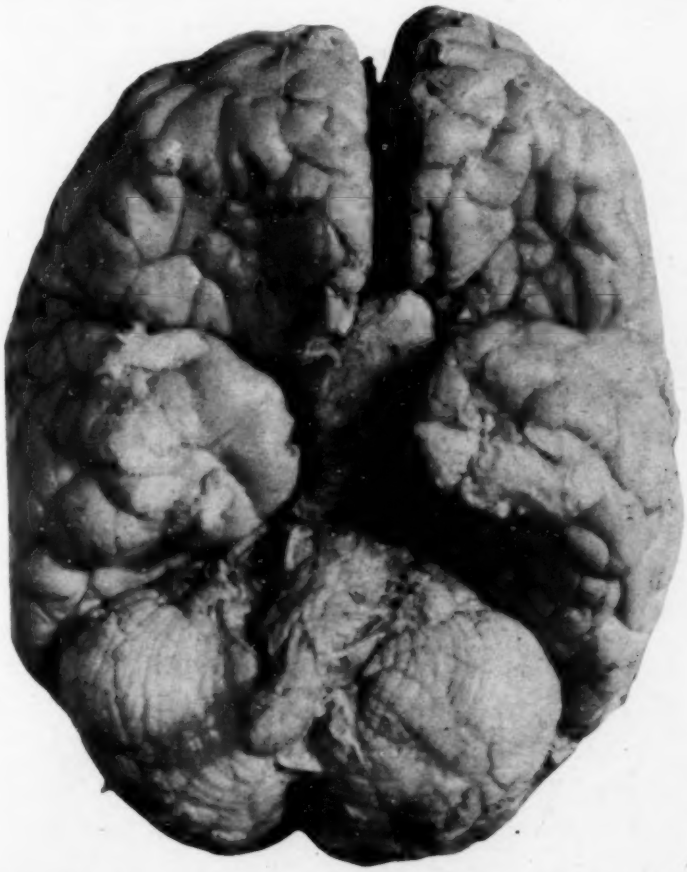


Fig. 11.—The location of the basilar tumor in the patient from whom the ventriculograms shown in figures 9 and 10 were taken.

is the result of the separation of the frontal and temporal horns by the tumor, the "brain swelling" on the side of the growth, and the dislocation of the homolateral frontal horn and body to the opposite side. In the other cerebral hemisphere, the body and frontal horn are pushed outward so that the vertical planes between the body and the anterior horn on the one hand and the temporal horn on the other are nearer together. These changes can sometimes be observed on the ventriculogram, and

they are never seen in cerebellar tumors. Therefore, if they do occur, they indicate that the presumed growth is not in the posterior cranial fossa.

To summarize the knowledge to be gained from ventriculograms in tumors of the posterior fossa, we would state that uniform dilatation of both the lateral and the third ventricles means an obstruction in the aqueduct or fourth ventricle. If, in addition, the ventriculograms show

Fig. 12.—Another view of tumor shown in figure 11 (sagittal section).

that the aqueduct and fourth ventricle are dilated, it is more probable that the lesion is not a new growth but is caused by some disease such as basilar meningitis or cisternal arachnoiditis which has interfered with the free exit of fluid from the fourth ventricle and the basal cisterns. The contralateral ventricle is usually larger than the homolateral one, and from this and from the outward, upward and forward dislocation of the occipital horn of one ventricle, one can often correctly diagnose the side on which an expanding subtentorial lesion occurs.

ESTIMATIONS OF AMOUNT OF FLUID IN LATERAL VENTRICLES FOR DIAGNOSIS OF CEREBELLAR LESIONS

By the expression "ventricular estimation," we ordinarily mean the information obtained by puncture of one or both lateral ventricles and from the evidence of changes in the position of the ventricles in the cerebral hemispheres, and the information concerning the amount of enlargement of the ventricular cavities based on the amount of fluid they contain, and the results of injections of colored dyes. In the follow-

Fig. 13.—Ventriculogram of the posterior horns of the lateral ventricles taken from the right in a patient with cerebellar tumor, showing that the right posterior horn *A* is displaced forward as compared with the left posterior horn *B*. *C* indicates the trephine opening.

ing paragraph, we shall refer mainly to the puncture of one or both lateral ventricles to determine the amount of fluid in the ventricular cavities.

As has already been mentioned, marked dilatation of the lateral ventricles with from 100 to 200 cc. or more of fluid occurs frequently in expanding lesions in the posterior cranial fossa which compress the

Fig. 14.—The right lateral ventricle from the same patient as shown in figure 13. The distance of the posterior horn from the surface of the skull as compared with figure 15 should be noted.

Fig. 15.—The left lateral ventricle of the same patient as shown in figures 13 and 14. The distance of the tip of the posterior horn from the surface of the skull should be compared with the same measurement in figure 14. That the ventricle is larger than that shown in figure 14 should be noted.

fourth ventricle or the aqueduct; but occasionally little hydrocephalus is present and only a small amount of fluid (10 or 30 cc.) can be obtained by puncture of one posterior horn. An index of the degree of dilatation may be obtained from the distance that the needle must be passed into the brain before fluid escapes. This is especially valuable if the body of a ventricle is punctured, and the ventricle is reached 1 or 2 cm. instead of 4 or 5 cm. from the surface.

The amount of fluid that escapes from the needle is not always an evidence of the amount of dilatation of the ventricle that was punctured; if the foramina of Monro are patent, much of the fluid may come from the other lateral ventricle which may be the more dilated of the two.

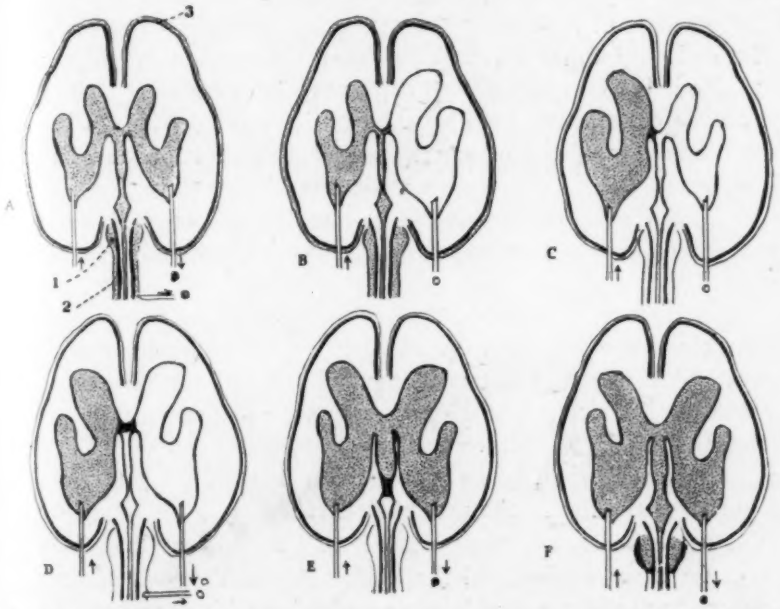


Fig. 16.—Diagrammatic illustration of the course of fluid injected into a lateral ventricle in ventricular blocks at various locations. In *A*, showing the normal ventricle, *1*, indicates the cisterna magna; *2*, the spinal subarachnoid space and *3*, the cerebral subarachnoid space. *B* shows a block of the contralateral foramen of Monro; *C*, a block of the homolateral foramen of Monro; *D*, a block of both foramina of Monro; *E*, block of iter and upper part of fourth ventricle and *F*, block of channels of exit from cisterna magna. The diagrams show the information gained by puncture of both lateral ventricles.

If more than 40 or 50 cc. is obtained, dilatation of at least one ventricle is present; in a patient with the symptoms and signs of a tumor in the posterior cranial fossa, amounts of 100 cc. or more mean obstructive hydrocephalus which probably is due to the subtentorial new growth. We say "probably," because in marked bilateral distended ventricles from any cause there may be signs of cerebellar disturbance from the hydrocephalus itself.

In patients with intracranial expanding lesions, in whom it is uncertain whether the growth is in one frontal lobe or in the cerebellum on the opposite side, the fact that the lateral ventricles contain an excess of fluid makes the subtentorial location of the growth probable. It is well to remember in this connection that the growth may be in neither of these locations but in the aqueduct of Sylvius. Recently, one of us explored the left frontal lobe for a suspected left frontal new growth. None was discovered, but the homolateral ventricle was found to contain a large amount of fluid which practically excluded a frontal new growth. A suboccipital exploration of the cerebellum was later performed in another neurosurgical clinic, also with a negative result. The patient finally succumbed, and postmortem examination disclosed a small growth in the aqueduct of Sylvius.

In order to draw conclusions from the amount of fluid in the ventricles it is often necessary to puncture both occipital horns before one can be certain that both lateral ventricles are dilated. Even then, from the results of the puncture alone, a conclusion cannot be drawn of the location of the obstruction in the ventricular pathways, and other procedures, which will be described later in the paper, will become necessary, unless the symptoms and physical signs point to subtentorial disease.

In obstructive hydrocephalus from tumors in or around the cerebellum, the pressure of the fluid within the ventricles is often but not always high. In many instances, the fluid spurts out of the needle in the ventricle for from 6 to 10 inches as soon as the ventricular cavity has been entered. If manometric tubes are connected with the needle, the pressure will often be so great (more than 50 cm.) that the fluid escapes from the top of the manometric tube.

On the other hand, the pressure may be so low that little fluid escapes from the needle, and the fluid, often in amounts of from 100 to 200 cc., has to be removed by slow aspiration with a syringe. In other words, the intraventricular pressure may be low with markedly dilated lateral ventricles and higher, with ventricles that are less distended. In some atrophic conditions of the cerebral hemispheres and in some pathologic processes whose nature is not yet understood, the intracranial pressure may be low, although the ventricles are markedly dilated, and 100 cc. or more of fluid can be withdrawn.

Thus, in a patient on whom one of us performed an exploratory operation with subtemporal decompression for suspected supratentorial disease, intracranial pressure was found to be below the normal, for the dura was not tense and the brain was found to lie at least 1 cm. away from the bone. When the ventricle was punctured, little fluid (under no pressure) escaped from the needle, but on aspiration, 120 cc. of fluid was easily obtained.

Another procedure may be of aid in the estimation of the amount of fluid in and of the degree of dilatation of both lateral ventricles. After a ventricular puncture has been performed and a large quantity of fluid has escaped from the needle, the veins of the neck are compressed by an assistant. If again a large quantity (from 50 to 100 cc. or more) of fluid runs out of the needle, it is probable that much of this second amount comes from the other lateral ventricle and that therefore the other lateral ventricle is also dilated and the foramina of Monro are patent. The compression of the veins of the neck (Queckenstedt procedure) is used most often in spinal lesions with level symptoms in order to determine whether a "spinal block" is present, but the procedure is also of value in studies and estimations of the amount of fluid in the lateral ventricles in subtentorial disease.

It is not within the scope of this paper to discuss the changes in the protein and sugar content of the ventricular fluid. We desire to mention, however, that if there is a doubt whether cerebellar symptoms are due to intrinsic vascular or degenerative disease or to a new growth, a high protein content is somewhat in favor of tumor.

Xanthochromia of the ventricular fluid usually means a supratentorial growth in or near the lateral ventricles. We have never observed yellow fluid in the lateral ventricles in tumors of the cerebellum or of the fourth ventricle or pons.

INTRAVENTRICULAR INJECTIONS OF DYES

There is a true circulation of the cerebrospinal fluid—the third circulation—and little of the fluid can leave the ventricular cavities by means of absorption or transudation through the ependymal lining of their walls. That the fluid finally again returns to the "mother liquor"—the blood—is certain, although the exact manner in which the fluid leaves the subarachnoid space is still a matter of mystery. These considerations have led to investigations of the patency of the ventricular pathways by means of injections of dyes into one part of the ventricular system and their recovery from other locations. If one or both foramina of Monro are occluded, the dye injected into one lateral ventricle cannot be recovered from the other. If the third ventricle, aqueduct or fourth ventricle are compressed, dye injected into a lateral ventricle cannot reach the spinal subarachnoid space, and finally, as the absorption of fluid from the ventricular walls is minimal, dye injected into a ventricular cavity enters the blood stream slowly and in only a small amount. Little can be found in the urine in the first few hours. Studies with dyes (fig. 15) are therefore of value in obstructive hydrocephalus caused by tumors or other expanding lesions in or about the cerebellum.

A variety of substances have been used for these studies—neutral phenolphthalein, neutral indigo carmine, methylene blue, weak tincture

of iodine, etc. If the procedure is to consist of the injection into one lateral ventricle and the attempted recovery of the dye from the other lateral ventricle or from the lumbar subarachnoid space, it is advisable to use a dye such as indigo carmine which can be recognized by its color without the additional use of an indicator. If, on the other hand, lumbar puncture cannot be done, because a lesion of the posterior fossa is suspected, and the fact of the retention of the cerebrospinal fluid within the ventricular system is to be determined by examination of the urine, then some substance such as phenolphthalein should be used, of which a definite amount is injected into a lateral ventricle. The urine is collected for two hours, and the amount of the dye in the urine determined by the usual colorimetric methods.[4]

In many instances, the injection of dye into one lateral ventricle gives only a limited amount of information, unless the other lateral ventricle is punctured at the same time.

If the clinical symptoms and signs point to the posterior fossa and the presence of a cerebellar neoplasm is most probable, the finding of a dilated ventricle by puncture of one posterior horn, followed by the injection of phenolphthalein into the ventricle and by examination of the urine which is secreted in the ensuing two horns, may give facts which support the diagnosis of an expanding lesion of the posterior fossa. If little of the phenolphthalein (from 0 to 5 per cent) appears in the urine during the period mentioned, it is probable that there is an obstruction in the iter or the fourth ventricle.

If, on the other hand, it has been impossible to determine whether a lesion is supratentorial or infratentorial, the posterior horns of both ventricles must be punctured, the amount of dilatation of each ventricle estimated from the amount of fluid withdrawn, and dye then injected through the one needle. The dye will promptly appear in the fluid from the other ventricle if the foramina of Monro are patent. If there is an obstruction in one or both of these foramina, the dye will not pass from the one into the other ventricle and the conclusion is justified that the neoplasm is located above the tentorium in a position in which it will directly obstruct or make pressure on the pathway between one or both lateral ventricles and the third ventricle. When this fact has been demonstrated, then lumbar puncture may be performed, and by injection of dye into each ventricle it is possible to determine whether one or both of the foramina of Monro are obstructed. This procedure is occasionally of great value both in order to exclude a lesion of the posterior fossa when both ventricles are dilated and in order to determine whether the obstruction is caudad or cephalad to the foramina of Monro.

4. This test cannot, obviously, be made unless the function of the kidneys is normal and other tests have demonstrated that renal disease is not present.

In almost all suboccipital craniotomies for suspected cerebellar tumors, the tension in the posterior cranial fossa must be diminished by puncture of one posterior horn and evacuation of fluid. In some of these cases, before the fluid had been evacuated, we have injected indigo carmine into the ventricle and have noted, after the dura had been opened, whether the fluid in the cisterna magna was or was not colored blue. If the fluid in the cistern remained uncolored, we were certain that the obstruction was cephalad to the foramina of Magendie and Luschka and at once looked further forward for the neoplasm. If, on the other hand, the fluid in the cisterna magna was colored blue, the growth had to be looked for below the fourth ventricle, and there was some probability that the lesion was not a tumor but an inflammatory process in the paths of exit of fluid from the basal cisterns.

SUMMARY AND CONCLUSIONS

In a large number of instances of cerebellar disease, the symptoms and signs of a lesion in the posterior cranial fossa are so well marked that studies of the ventricles are unnecessary. When the diagnosis of the location and of the nature of a disease are uncertain, the information to be obtained from studies of the ventricles is of no little value. Even then, however, the clinician must never forget that careful observation and thorough examinations are the efficient handmaidens of diagnosis, and that on them must rest the final burden of proof.

The evidence obtained by ventricular puncture and estimation, by the injection of dyes and by ventriculography is not always unequivocal, and one is sometimes dealing with probabilities rather than certainties. Tumors in one or the other location may cause pressure on more than one part of the pathways of the cerebrospinal fluid, and, as already mentioned, other parts of the ventricular system may be compressed by the dilated ventricles themselves.

The subject can perhaps be best summarized by a somewhat categorical statement of the questions which most often require an answer: 1. Is the disease in or around the cerebellum and vermis, and if so, is it a tumor? 2. Is the tumor located in one frontal lobe or in the cerebellar lobe of the opposite side? 3. Is the growth one in the posterior cranial fossa which has caused an obstructive hydrocephalus from pressure on the iter or the fourth ventricle, or is it supratentorial, with dilated ventricles because of pressure on the third ventricle?

1. If progressive signs and symptoms point to a cerebellar lesion, the presence of dilated ventricles determined by puncture of one or both posterior horns makes the presence of an expanding lesion probable. If, in addition, examination of the urine after injection of phenolphthalein into a lateral ventricle fails to show a large amount of the dye in the urine within two hours, the diagnosis of an expanding lesion in the

posterior cranial fossa is so well grounded that injections of air and ventriculography are unnecessary. The absence of bilateral ventricular dilatation does not entirely exclude an expanding lesion in the posterior cranial fossa, although it makes it improbable and makes the likelihood of a nonexpanding intrinsic cerebellar disease greater.

If, however, ventriculography has been performed, the occurrence of dilated ventricles with a regular outline gives support to the diagnosis of a lesion of the posterior fossa. The contralateral ventricle is usually the larger one, and the posterior horn of the lateral ventricle on the same side as the tumor is often dislocated forward, outward and upward.

2. When the diagnosis rests between a tumor of one frontal lobe or of the opposite cerebellar lobe, and puncture of the ventricle on the side of the possible frontal lesion discloses a dilated ventricle, the growth is, in all probability, in the posterior cranial fossa. If, in addition, the other lateral ventricle is punctured and is found to be also dilated and indigo carmine or other dye passes from one into the other lateral ventricle, the probability is made a certainty, and injections of air are not necessary. What has been mentioned under 1 is of course applicable also to this group of cases.

3. If the clinical diagnosis favors the presence of a supratentorial growth which is compressing the third ventricle but if a cerebellar neoplasm cannot be excluded, and puncture of the lateral ventricle on the side on which the tumor is presumed to be shows that the ventricle is dilated, a subtentorial lesion cannot be excluded until air has been injected and the ventriculograms studied. Tumors which cause a bilateral dilatation of the ventricles from compression of any part of the third ventricle usually cause some defect in the regular outline of the ventricles as seen on anteroposterior roentgenograms. If the ventriculograms show that the third ventricle is dilated, then the obstruction must be either in the hindmost part of the third ventricle or behind it, and the probability of a lesion of the posterior fossa becomes greater.

That there is a certain amount of danger from the injection of air by ventricular puncture is without question, and it would be a great advantage if, in lesions of the posterior fossa the air could with safety be injected by the lumbar route. Ventricular estimation should always be performed first; that is, a puncture of one or both lateral ventricles through their occipital horns in order to determine by the methods described in the body of this paper whether both lateral ventricles are dilated and the foramina of Monro are patent. In the majority of cerebellar tumors, ventricular puncture alone or ventricular estimation with studies by means of the injection of dyes will suffice, and injections of air and ventriculography will be performed only when the clinical and ventricular evidence of a cerebellar new growth is inconclusive.

CEREBELLAR TUMORS OCCURRING IN IDENTICAL TWINS*

FREDERIC H. LEAVITT, M.D.
PHILADELPHIA

This presentation is of interest because of the occurrence of cerebellar tumors in twins that were apparently of the monozygotic or identical type. The illustrations are those of tissue from the first twin who died. It was impossible to obtain the consent of the parents for the necropsy on the second twin. However, there is little doubt that the diagnosis of the lesion in the second twin was cerebellar tumor. The exact location and histopathologic type of this tumor is, of course, not known.

The first twin died in December, 1924, at the age of 6½ years. The second died in March, 1927, when 8½ years of age. At the time of the death of the first twin the second had not shown any symptoms of the illness; because of this, it was impossible to obtain measurements of the twin, particularly the patterns of their palms and soles.

My reason for believing that these children were of the monozygotic type was because of their striking similarity. The boys were born at term without artificial aid; both weighed 5¼ pounds (2.4 Kg.); they were so identical in appearance that it was necessary to tie a ribbon around the wrist of John in order to differentiate him from his brother Jules. The similarity as they grew up through babyhood was striking. Both had dark brown curly hair, dark brown eyes, and dark olive skin, and they were of the same size and stature. The boys began to walk at the age of 12 months and to talk at 14 months; they showed the same mental, physical and emotional characteristics. Both were fond of the same sort of food, and exhibited an extraordinary eagerness to play with fire. The mother said that they were much more mischievous and prone to get into trouble than any of her other children. When they were 2 years of age, Jules developed bronchopneumonia; following this, his physical growth was retarded and John began to outstrip him in height and weight and to become more robust.

REPORT OF CASES

CASE 1.—Jules was taken sick in October, 1924, with headache, which gradually increased in severity and frequency; this was soon followed by projectile vomiting and disturbance in walking. In the latter part of November he was taken to the Children's Hospital in Philadelphia, where he had several generalized convulsions and, where he died on Christmas Day, 1924. Ophthalmologic

* Read at the Fifty-Third Annual Meeting of the American Neurological Association, Atlantic City, N. J., May 25, 1927.

examination at the hospital revealed bilateral choked disk with an elevation of from 3 to 4 diopters above the retinal level. The eyeballs were parallel and there were no apparent extra-ocular palsies. A lumbar puncture was performed without untoward results; the routine examination of the spinal fluid did not reveal any abnormalities.

Postmortem Examination.—At autopsy, a tumor was found in the vermis of the cerebellum. When the brain was cut into sections after hardening, the tumor was found to measure approximately 5 cm. in diameter; it was of spongy consistency, not definitely infiltrating in character and apparently arose from the

1.—Photograph of Jules (the smaller) and John, showing the striking similarity in appearance.

region of the roof of the fourth ventricle. Examination of the ventricles did not show any enlargement as from a hydrocephalus, and the other tissues of the brain were found to be normal. The meninges were not involved.

Microscopic Study.—Report of the microscopic observations made at the laboratory of the Philadelphia General Hospital, as submitted by Dr. N. W. Winkelman, was as follows:

"The tumor comes under the general heading of gliomas. The types of cells present vary, but for the most part they fit in with what has been described by Bailey and Cushing as 'medullo-blastomas,' but other glial type

cells are present as is common in this type of tumor. Neuroblasts and spongioblasts are present to a small degree. The connective tissue stroma, which has a tendency to divide the tumor up into small islets, is a little more abundant than in the usual blastoma."

CASE 2.—John, the more robust of the twins, following Jules' attack of pneumonia, became sick in September, 1926, with gradually increasing headache, projectile vomiting and disturbance in walking, exactly the same symptoms that the mother had noted in John. The boy would suddenly put both hands to his head and scream with pain. This act would frequently be followed by sudden projectile vomiting without nausea. He soon developed a distinctively cerebellar gait, and became constipated. About Christmas Day, 1926, he had a series of generalized convulsions lasting several hours; after these attacks

2.—Sagittal section showing the large infiltrating glioma arising from the roof of the fourth ventricle.

he was unable to walk and was confined to bed. Subsequently, he became totally blind in the right eye, and the vision of the left eye was limited to perception of light. He developed skew deviation of the eyeballs and bilateral deafness. The opthalmologic report of Dr. Warren Reese showed 4 diopters of choking in the right disk and 3 diopters in the left disk, with marked blurring of each. The family would not permit the child to be taken to a hospital, and he died at home in March, 1927.

A review of the literature has failed to reveal any recorded instance of cerebellar tumors occurring in identical twins. Harvey Cushing [1]

1. Cushing, Harvey: The Intracranial Tumors of Preadolescence, Am. J. Dis. Child. **33**:551 (April) 1927.

makes note of the relative predominance of cerebellar tumors over that of cerebral tumors in children, the proportion being about two to one, whereas in adults it is about one to five. Cushing and Bailey [2] state: "The medulloblastomas are essentially tumors of children and usually occur in the midcerebellar region arising from the roof of the fourth ventricle. They are rapidly growing tumors and commonly arrive in the worst possible places to endanger life." In a review of a great number of instances of this type, Cushing states that the progress of the condition in these children is rapid, and that convulsions frequently occur with symptoms of internal hydrocephalus. He observed that the ataxia is more evident in the lower extremities than in the upper, and frequently a history is obtained of cerebellar seizures with retraction of the head, respiratory difficulty and other symptoms of basilar meningitis. Nystagmus has been an inconspicuous feature of the cerebellar syndrome; extra-ocular paralyses are common. The average age of the patients is 10 years. Cushing and Bailey further state that:

> The tumor seems to have its origin over the roof of the fourth ventricle. It is well known that the cells forming the cerebellum migrate in an extraordinary way during embryonic life, and it would seem to be not improbable that in this situation groups of cells might become isolated and retarded in their development. Should these isolated cellular groups differentiate, heterotopic malformation would result. Pfleger found such groups in the region of the fourth ventricle in 75 of 400 supposedly normal cerebella. If such an isolated and retarded group of cells should undergo a neoplastic transformation, such a tumor as we have described might be expected to result. The majority of neoplastic cells are potentially neuroglia cells, some are potential neurones, and it is on this basis that the designation of medulloblastomas has been adopted. They are in reality composed of embryonic medullary cells.

Newman [3] states that if twins are alike structurally, they must be alike functionally. A pair of twins with identical brains should have identical mental development at birth, but further mental development depends on training and environment. There is remarkable unity of thought and action in duplicate twins.

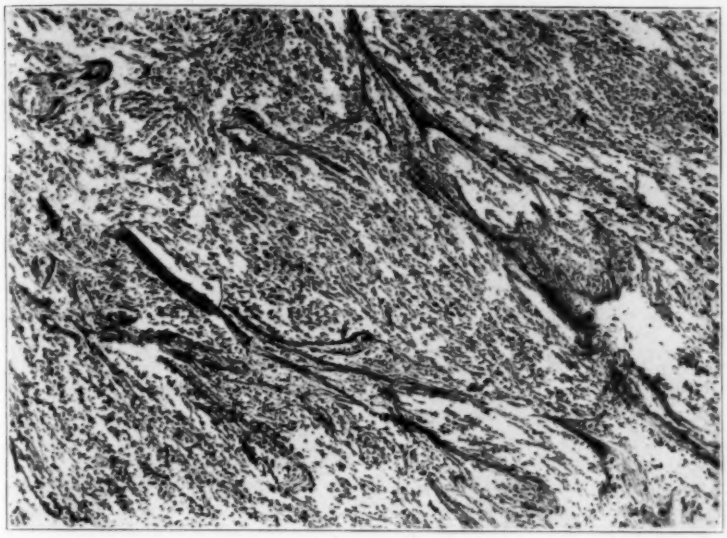
Mitchell [4] reported twenty-four instances of the occurrence of mongolian idiocy in identical twins. His conclusions are that mongolism is a defect of the germ plasm and is against any theory that factors

---

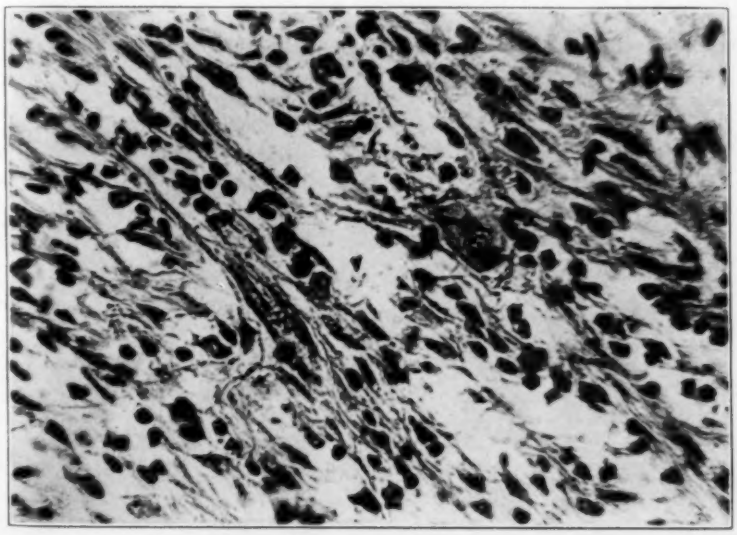
2. Bailey, Percival; and Cushing, Harvey: Medulloblastoma Cerebelli Common Type of Midcerebellar Glioma of Childhood, Arch. Neurol. & Psychiat. **14**:192 (Aug.) 1925.

3. Newman, Horatio Hackett: The Biology of Twins, Chicago, The University of Chicago Press, 1917.

4. Mitchell, A. Graeme, and Downing, Harold F.: Mongolian Idiocy in Twins, Am. J. M. Sc. **172**:866 (July-Dec.) 1926.



3.—High power view showing the stroma of connective tissue with a tendency t divide the tumor up into islets.



4.—High power view showing the typical structure of medulloblastoma. Glial type cells, neuroblasts, and spongioblasts are present in the connective tissue stroma.

operative during pregnancy are at fault. Mongolism occurring in one of twins, the result of a single ovum pregnancy, has never been demonstrated.

Abt,[5] in an article on "Diseases and Fate of Twins," cites many instances of abnormalities, deformities, diseases and defects in similar and dissimilar twins, but does not make any mention of identical cerebellar tumors. Murray,[6] in his treatise on "Twins in Health and Disease," does not make any mention of this condition.

CONCLUSION

I believe that the identical occurrence of cerebellar tumors in monozygotic twins may be construed as evidence strongly supporting the "fetal rest" theory of the development of the neoplastic tissue. There can be little doubt that the twins had cerebellar neoplasms in similar situations, and, as the structure of the tumor examined was composed of embryonic cells, and, as it is believed that monozygotic twins develop as a result of the splitting of a single fertilized ovum, I am of the opinion that both of these tumors were due to a "fetal rest" of embryonic tissue remaining as a pathologic nidus in the roof of the fourth ventricle of each twin, remaining there in a quiescent state from the time of the splitting of the original single ovum until some years later when some factor stimulated the pathologic growth of this group of embryonic cells in each twin.

ABSTRACT OF DISCUSSION

DR. PERCIVAL BAILEY: The occurrence of this type of cerebellar tumor is an extraordinary phenomenon which I have not seen before. Dr. Leavitt's explanation seems logical. The microscopic anatomy is consistent with medulloblastoma. Overgrowth of connective tissue forming a stroma is occasionally seen in these tumors, although I know of only two overgrowths such as seem to exist in this case. The diagnosis in these cases seems to be unmistakable.

---

5. Abt, Isaac A.: Diseases and Fate of Twins, J. Iowa State M. Soc. **14**:395 (Sept.) 1924.

6. Murray, George R.: Twins in Health and Disease, Lancet **1**:529 (March 14) 1925.

SUBACUTE DIFFUSE EPENDYMITIS

REPORT OF A CASE SIMULATING TUMOR OF THE BRAIN *

JOSEPH H. GLOBUS, M.D.

AND

ISRAEL STRAUSS, M.D.

NEW YORK

Two studies, one by Walther [1] and the other by Mérle,[2] stand out prominently among the contributions to the subject of ependymitis. In the first, the author draws attention to the so-called acute purulent ependymitis. He regards it as rather frequent in association with a coexistent inflammatory process elsewhere in the brain or meninges, and rare as an independent or definitely local inflammatory lesion in the ventricular lining of the brain. Without establishing any definite relationship between it and the more common, so-called chronic, form of ependymitis, he opens the discussion of the second form by subgrouping it into two types: (1) chronic diffuse sclerosing ependymitis, and (2) chronic granular ependymitis. He emphasizes the lack of sharp lines of division between these two types, since there are many transitional forms. In diffuse ependymal sclerosis, the ependyma is thickened, sclerosed over extensive areas and presents a smooth, though somewhat opaque surface; the second type reveals a more characteristic picture, the ventricular lining being studded with numerous elevations, the so-called ependymal granulations. The latter form of ependymitis has been much studied, but its histogenesis is still not clearly understood. While it is quite characteristic of the lining of the fourth ventricle in general paralysis, it nevertheless cannot be regarded as pathognomonic of that disease, since it is found, though less frequently, in other conditions such as tuberous sclerosis, senile dementia, cerebral atherosclerosis, multiple sclerosis, idiopathic chronic internal hydrocephalus, essential epilepsy and even in dementia praecox (Thurwachter [3]).

---

* Read at the Fifty-Third Annual Meeting of the American Neurological Association, Atlantic City, N. J., May, 1927.

* From the Neuropathological Laboratory and Neurological Service, Mount Sinai Hospital, New York.

* Aided by a grant from the Emanuel Libman Fellowship Fund.

1. Walther, Karl: Ueber chronische und akute Ependymentzündungen der Grosshirnventrikel, Inaugural Dissertation, Wiesbaden, 1897.

2. Mérle, Pierre: Étude sur les ependymites cérébrales. Thèse pour le doctorat en médecine, Paris, 1910.

3. Thurwachter, Ludwig: Ueber die diagnostische Bedeutung der Ependymitis granularis, Inaugural Dissertation, Leipzig, 1909.

In a more recent contribution, Mérle reviews the entire subject of ependymitis and makes some new observations. Of interest here, however, are those dealing with the acute and subacute forms. He also finds that acute purulent ependymitis occurs frequently in association with coexisting inflammatory processes of the brain, particularly in the various forms of purulent meningitis and of abscesses in proximity to the ventricular cavity. It is also his opinion that, while it is rare, acute ependymitis does occur as a primary and distinctly localized condition. He reports some experimental observations in support of this view. He is inclined to agree with Nouss [4] that in such instances the choroid plexus is often the channel of infection permitting the filtration of an organism, or else the choroid plexus serves as a bridge to transmit the inflammatory process from the meninges to the ependymal lining. He recognizes that instances of isolated ependymal inflammation are recorded in which the choroid plexus and the meninges failed to reveal pathologic changes. Thus, the question as to the channel of infection in acute ependymitis still remains open, though it seems that, for the present, an extension of a meningeal process will have to be accepted as the most probable common cause of ependymitis.

As Mérle has already pointed out, acute ependymitis is not always a terminal event. It may pass into a subacute inflammatory stage, as in his own and several other reported cases of ependymal involvement in which subacute productive changes were revealed. These were characterized by alterations in the ependymal lining with areas of desquamation alternating with areas of swelling and proliferation in the epithelial lining as well as with marked gliosis and perivascular infiltrations in the subependymal zone. These alterations in the ependyma not infrequently resulted in formation of a scar with partial or complete obliteration of normal communicating channels in the ventricular system, with occlusions occurring most frequently in the aqueduct of Sylvius, in the cavity of the fourth ventricle and in the foramina of Monro, where obstruction to the normal flow of cerebrospinal fluid results most effectively in the formation of various forms of internal hydrocephalus. Bilateral symmetrical hydrocephalus is caused by atresia of the aqueduct of Sylvius, as in the cases reported by Schlapp,[5] by closure of the fourth ventricle, as in the case of Tillgren,[6] and unilateral internal hydrocephalus by closure of the foramen of Monroe, as reported by Spiller [7] and

4. Nouss: Hydrocephalus internus acutus, quoted by Mérle (footnote 2).

5. Schlapp, M. G., and Gere, B.: Occlusions of the Aqueduct of Sylvius in Relation to Internal Hydrocephalus, Am. J. Dis. Child. **13**:461 (June) 1917.

6. Tillgren, N.: Ein seltener Fall von Ependymitis des IV Ventrikels, Ztschr. f. klin. Med. **63**:153, 1907.

7. Spiller, W. G.: Two Cases of Partial Internal Hydrocephalus from Closure of the Interventricular Passages, Am. J. M. Sc. **124**:44, 1902.

Cushing.[8] Some of these obstructive phenomena, which are due to scar formation in the interventricular channels, may occasionally give rise to a clinical picture not unlike that of cerebral neoplasm. They are often included in the vague group of pseudotumor cerebri, a term suggested by Nonne,[9] and which includes a large assortment of pathologic conditions.

The case reported here could also be designated as pseudotumor cerebri, since the diffuse ependymitis resulted in the formation of a structure which, by its location, gave rise to clinical manifestations that made the diagnosis of cerebral neoplasm most probable, and, in fact, excluded any other diagnosis except that of internal hydrocephalus due to obstruction in some part of the ventricular system. But the present tendency is to avoid the use of this term, since pseudotumor cerebri does not have an anatomic basis. The occurrence of a large variety of lesions in the brain which may, during the clinical course, display features simulating tumor of the brain is not sufficient to justify the use of such a term.

This view finds support in many contributions on the pathology of the so-called pseudotumor cerebri, notably by Bailey,[10] Finkelburg and Eshbaum,[11] Finkelburg,[12] Marinesco and Goldstein,[13] Mocquin,[14] Raymond[15] and Rosenthal,[16] as also in the anatomic observations in our case.

REPORT OF CASE

History.—A. Z., aged 42, married, a salesman, was admitted to the Mount Sinai Hospital on Oct. 7, 1925, complaining of headache, vomiting, dizziness and staggering gait. With the exception of an attack of jaundice thirty years before,

8. Cushing, H. in Keen: Surgery: Its Principles and Practice, Philadelphia, W. B. Saunders Company, 1919, vol. 3.

9. Nonne, M.: Ueber Fälle von Symptomcomplex Tumor Cerebri mit Ausgang in Heilung, Deutsche. Ztschr. f. Nervenh. **27**:169, 1904.

10. Bailey, Percival: Contribution to the Histopathology of Pseudotumor Cerebri, Arch. Neurol. & Psychiat. **4**:401 (Oct.) 1920.

11. Finkelburg and Eshbaum: Zur Kenntniss des sogenannte Pseudotumor Cerebri mit anatomischem Befund, Deutsche Ztschr. f. Nervenh. **38**:35, 1910.

12. Finkelburg: Ueber Pseudotumor Cerebri, Zentralbl. f. d. ges. Chir. u. i. Grenzgeb. **15**:515, 1912.

13. Marinesco, G., and Goldstein, M.: Deux cas de pseudo-tumeur cérébrale; Meningite sereuse et hydrocéphalie acquise. Extrait de la nouv. Iconog. de la Salpêtrière, no. 1, Jan.-Feb., 1912; quoted by Mérle (footnote 2).

14. Mocquin: Pseudo-tumeur cérébrale par empyème ventriculaire.: Nouv. iconog. de la Salpêtrière, 1905; quoted by Mérle (footnote 2).

15. Raymond, F.: Pseudo-tumeurs cérébrales et meningite sereuse ventriculaire, Presse méd. **18**:205, 1910.

16. Rosenthal: Histologische Befunde bei sogenannte Pseudotumor Cerebri, Ztschr. f. d. ges. Neurol. u. Psychiat. **7**:163, 1911.

he had always been well. On April, 1924, seven months prior to admission to the hospital, he began to suffer with frequent attacks of dizziness which would occur almost daily and would last for an hour or more; they were often followed by projectile vomiting. At the same time, the gait became unsteady. Competent neurologists found that he had bilateral papilledema, diminished knee jerks and an unsteady gait. The cerebrospinal fluid was under reduced pressure, and very little fluid was obtained on puncture; after the initial, rather meager flow, it soon stopped running. The Wassermann tests of both the spinal fluid and the blood were negative. Except for mild headaches, the status remained unchanged until about Aug. 20, 1924, when singultus of a rather persistent character set in. He then reentered the Neurological Institute, where, on August 26, ventriculography was performed. At this time, he manifested mild psychic disturbances, such as clouded orientation and impairment of memory for recent events. There was also some inequality of reflexes; the right knee jerk was greater than the left, and the right abdominal reflexes were less active than the left; pathologic reflexes were not present. At a somewhat later date (September 25) there was distinct impairment of the sense of smell on the left side.

About this time the report of a roentgenographic examination was: "There is evidence of increased intracranial pressure with the probability of a lesion to the left of the middle line and anteriorly placed." The headache had become more intense and more frequent. It would begin in the morning, continue for several hours and disappear, returning later in the day. The pain in the head would begin at the vertex and radiate forward in the direction of the left frontal region, where it would reach its highest intensity. Vomiting would usually set in an hour later. Recently he had also developed weakness in the lower extremities, and it had become more difficult for him to get around without aid or support.

Examination.—The patient was well developed and well nourished; there was percussion tenderness over the frontal region; the pupils were small but regular and reacted well to light and in accommodation. Nystagmus was not present; the ocular muscles were intact. The ophthalmologic report was: "Perivascular exudates, several retinal hemorrhages, mainly in the left eye, patches of retinal edema, intense bilateral papilledema. Visual acuity: right eye, 20/50; left eye, 20/200. The visual fields show slight uniform contraction which is a little more marked in the right than in the left eye."

There was slight weakness of the left lower extremity. Ataxia and disturbance in check phenomena were not noted. The reflexes in the left arm were more active than those in the right, and those of the right leg were greater than those in the left. The cardiovascular, respiratory and genito-urinary system were normal. The blood pressure was: systolic 94 and diastolic 76. Urinalysis and examination of the blood gave negative results.

Course.—On Oct. 24, 1924, indigocarmine was injected into the ventricles by Dr. Elsberg. A trephine opening was made over the left posterior horn; a needle was inserted, and from 40 to 50 cc. of clear fluid was removed; 1 cc. of indigocarmine was injected. A needle was then put into the right ventricle through an old trephine opening, and clear fluid was obtained, indigocarmine not passing from the left into the right ventricle. Lumbar puncture was performed twenty minutes later, and the results showed that indigocarmine did not pass into the lumbar fluid, which was slightly bloody. Previous ventriculograms showed that air would not pass from the right into the left ventricle, and that air would not enter the third ventricle. The indigocarmine injection showed that dye would not pass from the left into the right ventricle. As both ventricles were dilated, the conclusion was reached that both foramina of Monro were obstructed.

On Dec. 1, 1924, an ophthalmologic examination revealed: right optic disk swollen, with small hemorrhage; physiologic pit absent; veins dilated and thrombosed; margin covered by connective tissue; arteries small and tortuous. Adjacent to the disk there were small glistening spots. In the left eye a similar condition to that in the right eye, but more advanced, was found. This was interpreted as progression of pathologic changes in both eyes.

A neurologic examination at this time disclosed: slight central facial weakness on the right side; slight inequality of abdominal reflexes; knee jerks diminished and unequally active; ankle jerks active and equal; bilateral pseudoclonus; no pathologic reflexes; no impairment of the sense of smell; slow cerebration. The fundal changes, the clinical picture and the mental changes suggested the diagnosis of a cerebral neoplasm in the middle fossa and between the hemispheres.

On Dec. 10, 1924, the fundi showed recession of the papilledema and formation of new blood vessels. The gait was unsteady and guarded, the trunk held stiffly, the head inclined to the right, the right shoulder higher than the left, the trunk tilted to the left. The patient favored the right leg, bringing it up slowly and not with the same facility as the left. Definite adiadokokinesis and past pointing were not present.

A tumor in the third ventricle was regarded as the most probable diagnosis. On Dec. 13, 1924, the patient was allowed to go home, as a mild recession in the symptoms warranted a delay in operative intervention.

The patient was readmitted to the hospital on Feb. 2, 1925, complaining of frontal headache, frequent vomiting, unsteady gait with a tendency to fall to the right and of urinary incontinence. On admission, he was dull, poorly oriented, walked with difficulty and unsteadily on a broad base with marked uncertainty in step, with head tilted to the right and diminished associated movement in the right arm. He dragged the right leg. There was mild bilateral papilledema, with nystagmoid movements in both lateral planes and limitation of external rotation of the left eye. The pupils were irregular, small and reacted sluggishly to light and in accommodation. There were: slight weakness of the right side of the face; weakness in both lower extremities; diminished abdominal reflexes and knee jerks; active achilles jerks and no pathologic reflexes. The posture of the head, tendency to fall to the right and the cerebellar gait suggested the possibility of a neoplasm of the posterior fossa. A caloric test gave, however, normal responses. The visual acuity had declined: right eye, 12/200; left eye, 12/200.

On Feb. 16, 1925, a third ventriculography was performed by Dr. Elsberg, who reported that the right ventricle was easily reached at a depth of 3 cm. by means of a needle which was passed through the old scar and trephine opening. The posterior horn was entered, 30 cc. of clear fluid was removed, and 35 cc. of air was injected. Roentgen-ray examination showed marked enlargement of the anterior horn. The patient was apparently in good condition for fourteen hours after the injection of air, which was left in the ventricle, although he was somewhat drowsy.

Two days later, however, the patient began to grunt and passed into a comatose condition. The right arm displayed moderate rigidity, and bloody fluid was withdrawn from both ventricles. Ten minutes later, the patient had a severe convulsion involving first the right side of the body, then the head; the eyes were turned to the right; the convulsion then spread to the left arm and leg. During the convulsions, the patient was deeply cyanosed, became unconscious and died within a short time. Only during the agonal period was there a rise in temperature.

SUMMARY AND COMMENT

The outstanding clinical features in the case were the manifestations of increased intracranial tension, including almost all of the cardinal signs and symptoms of cerebral neoplasm, such as attacks of dizziness, intense headache, projectile vomiting and papilledema. These signs and symptoms, with the gradual unfolding of the clinical picture, interrupted by slight remissions, and the absence of febrile reaction justified a diagnosis of tumor cerebri. The absence of well defined focal signs, however, made localization of the lesion difficult. A median line location, possibly between the hemispheres, was suggested by the bilaterality of symptoms. The ventriculographic studies and the injections of dye, however, were of distinct service. They revealed the presence of bilateral, symmetrical dilatation of the lateral ventricles and demonstrated conclusively the lack of intercommunication between the lateral ventricles of the two hemispheres owing to closure of the foramina of Monro, with consequent lack of communication between the third and the lateral ventricles. These observations definitely localized the lesion and placed it in the third ventricle. The true nature of the process of the disease was not recognized during the life of the patient.

With the observations at necropsy in this case, the nature of the lesion being clearly understood, it can be said that, given a similar case with exactly the same clinical course and observations, the only diagnosis possible would be the one made, for there are no means of identifying, during the life of the patient, the pathologic process to be described.

Necropsy Findings.—Gross Anatomy: The brain was removed in two segments through an enlargement of the small trephine openings. Before removal, the ventricles were accidentally punctured and a large amount of somewhat blood-tinged cerebrospinal fluid escaped. The surface of the brain showed evidence of marked intracranial pressure. The pia-arachnoid was glistening and filled with a somewhat opaque fluid. The right cerebral hemisphere was removed first; the lateral ventricle was markedly dilated in all subdivisions. The foramen of Monro could not at first be located, but on careful search a communicating opening between the third and lateral ventricle was found, which was covered and almost closed by the swollen and markedly thickened ependyma. The wall of the third ventricle showed normal markings but was found to be depressed and stripped of its ependymal lining. The choroid plexus of the lateral ventricle was cystic and voluminous. Later examination of this hemisphere, after fixation in formaldehyde, showed that the ependymal lining wall of all subdivisions of the lateral ventricle was markedly thickened, streaked with hemorrhagic foci and stripped easily from the underlying subependymal structure. The corpus callosum was markedly thinned and represented by a narrow zone of white matter. The course of the ventricular puncture could be followed easily but a particular reaction in the lining membrane of that region in the posterior horn of the lateral ventricle was not found. Cutting horizontally at various levels did not reveal neoplastic growth anywhere in the substance of this hemisphere.

The left hemisphere showed a similar dilatation of the lateral ventricle in all parts with a similar thickening of the ependymal lining and a similar increase in volume and cystic appearance of the choroid plexus. Here again the foramen of Monro could not be seen because the opening was closed by thickened ependyma. The mesial surface of the left optic thalamus showed a marked concavity due to compression by a cystic structure. This sac, which was membranous, was strikingly similar to the ependymal lining of the ventricular cavity and was funnel-shaped with its apex inserted into the aqueduct of Sylvius (fig. 1). It was now partially collapsed and appeared to represent a cyst, but was later proved by microscopic examination to be thickened ependyma formed into a sac which filled the third ventricle during life. Search for neoplastic tissue failed to disclose any tumorous growth. In the fourth ventricle, the ependymal lining was also markedly swollen and narrowed down at its posterior end, so that it probably occluded the central canal. Neoplastic tissue was not found in or about the fourth

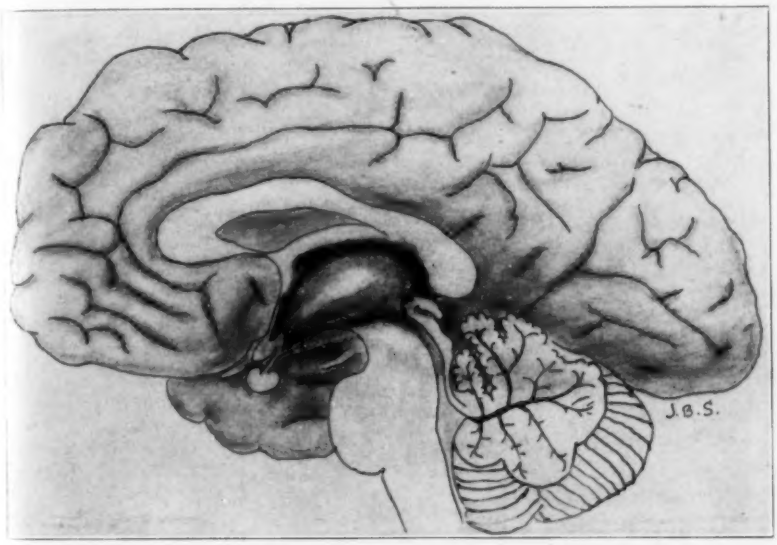


Fig. 1.—Drawing of the mesial surface of the hemisphere illustrating the depth of the third ventricle, the contained pseudocyst and the small funnel-shaped extension into the aqueduct of Sylvius.

ventricle. The provisional anatomic diagnosis was: chronic ependymitis, producing a pseudocyst formation in the third ventricle with closure of the foramina of Monro and of the aqueduct of Sylvius.

Microscopic Anatomy: The histologic observations are based on a study of blocks of tissue from various regions: the several lobes of each hemisphere with the cortex and subcortex; the meninges; the ependymal lining of all subdivisions of the ventricular cavities, and, particularly, the ependymal lining of the aqueduct of Sylvius and of the fourth ventricle, as well as the wall of the "pseudocyst" in the third ventricle.

The Cerebral Hemispheres: Striking changes were not found in the cortex; the general architecture was intact, the ectodermal components, aside from a mild increase in the glia, did not show any changes; the mesodermal components were also free from morphologic changes. The subcortex, however, showed well

defined diffuse alterations in marked increase in the glia, which manifested itself in advanced mobilization of glia cells about medium sized blood vessels; in this the oligodendroglia cells played a dominant rôle. As the subcortex approached the ependymal lining, it displayed a more pronounced gliosis. With this increase in proliferation of the glia cells there appeared a more pronounced picture of inflammatory reaction in the nature of adventitial infiltrations about the smaller and medium size vessels (fig. 2), consisting of small and medium size lymphocytes, polymorphonuclear neutrophils and occasional fibroblasts. Still more definite

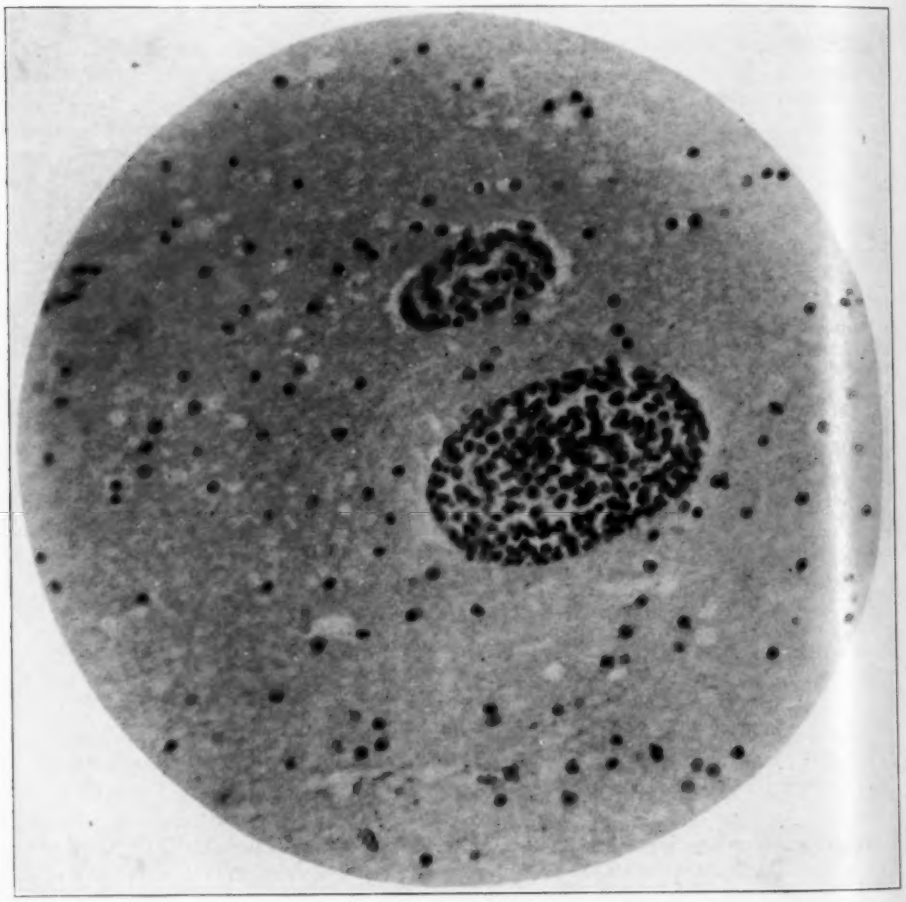


Fig. 2.—A typical area of the periependymal subcortex, showing vessels with adventitial infiltrations.

changes were found in the ependymal lining and in the subependymal zone, which varied with the distance of the ventricular subdivision from the third ventricle, the seat of the main pathologic process. They were mild in the more remote portions of the lateral ventricles, became more pronounced as they approached the foramina of Monro and reached the height of development in and about the third ventricle. They gradually declined as they passed on posteriorly through the aqueduct of Sylvius into the fourth ventricle. In areas roughly corresponding to

the anterior horns of the lateral ventricles, there was still a fairly well preserved ependymal lining (fig. 3), though definite inflammatory changes were present in the subependymal zone, such as mild gliosis in the subependyma with adventitial infiltrations of the blood vessels. Changes similar in degree were also found in the descending horns. As the foramina of Monro were approached, the pathologic changes became more pronounced; the ventricular lining was denuded of its ependymal coat, the latter having been replaced by a narrow zone of homogenized material which, in its turn, was surrounded by a somewhat wider zone of gliosis

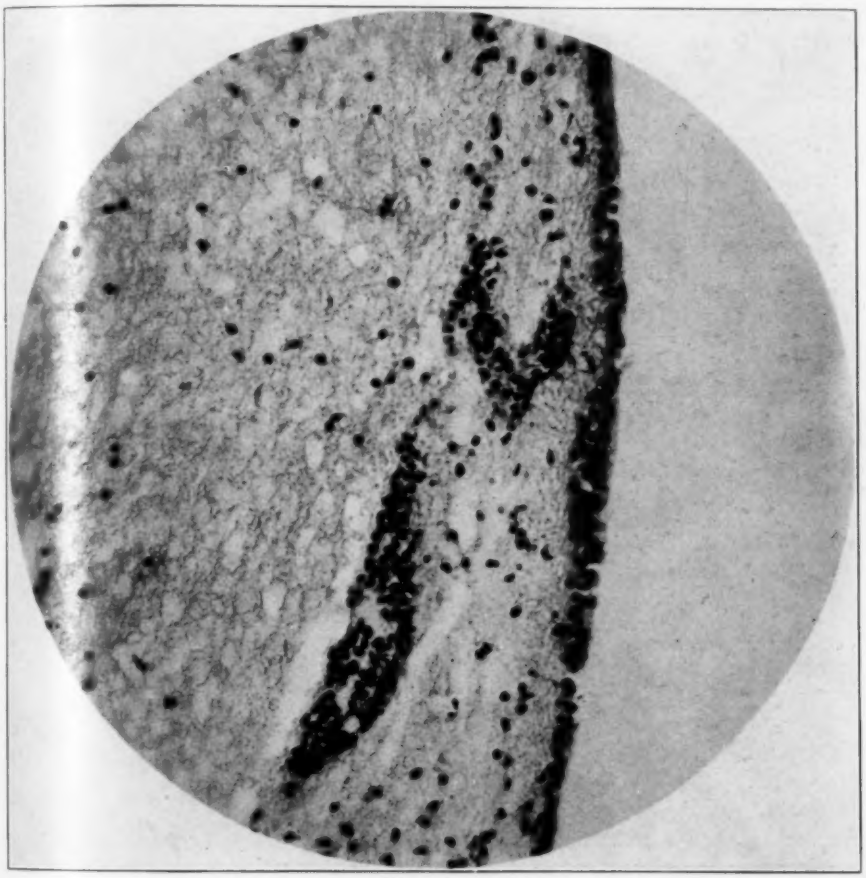


Fig. 3.—Section illustrating intact ependymal lining of the anterior horn of the lateral ventricle, with inflammatory changes in the subependyma.

and inflammatory mesodermal reactions. Here, more numerous vessels showed adventitial infiltrations (fig. 4).

In the ependymal lining of the aqueduct of Sylvius changes of a more chronic character were found; it was entirely disorganized and was replaced by a wide zone of homogenized necrotic tissue giving the impression of a hyalinized band distinctly separated from the underlying subependyma. The latter showed a wide zone of gliosis (fig. 5). Through the aqueduct of Sylvius as well as in the floor of the fourth ventricle, there were numerous elevations of the ependyma, grossly

simulating the so-called ependymal granulations. The histologic appearance of these granulations was that of rounded protuberances of the necrotic ependyma supported by local increase in the subependymal glia (fig. 6). The subependyma here presented evidence of a more chronic productive inflammatory process: glial fibrosis and marked fibrosis of the adventitial coat of the blood vessels.

The Pseudocyst: The cystlike structure revealed little more than thickened subependyma with a narrow zone of necrotized ependymal coat (fig. 7). There

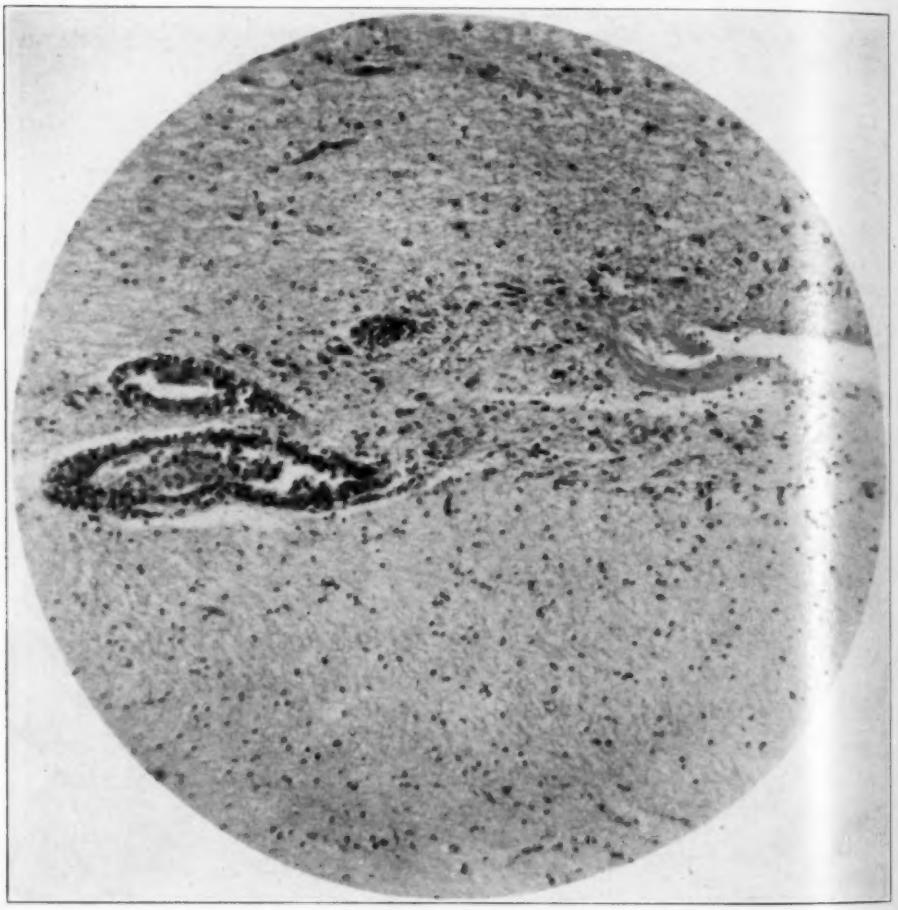


Fig. 4.—Section showing the appearance of the ependymal zone near the foramen of Monro. The epithelial lining is lost and replaced by a necrotic band of tissue. The subependyma shows fairly active gliosis and vessels with adventitial infiltration.

were many local variations in its wall. In some areas, as in figure 7, a small amount of brain tissue was still adherent to the subependymal zone; the latter showed numerous infiltrated vessels, a relative increase in neuroglia and many wandering lymphocytes free in the tissue. The ependymal lining was completely devoid of its normal structure and was replaced by a narrow band of homogenized necrotic tissue. In other areas, the ependyma and subependyma were replaced

by a wider band of necrotic tissue, interrupted in places, overlying a wider zone of gliosis in the subependymal region. The latter showed many wandering lymphocytic elements in addition to the glial proliferation.

The Meninges and Choroid Plexus: The meninges did not show any striking changes aside from occasional areas of thickening, particularly in the interpeduncular space, due to mild inflammatory reaction. The choroid plexus presented large accumulations of fat in the pendymal cells by the Marchii stain, but

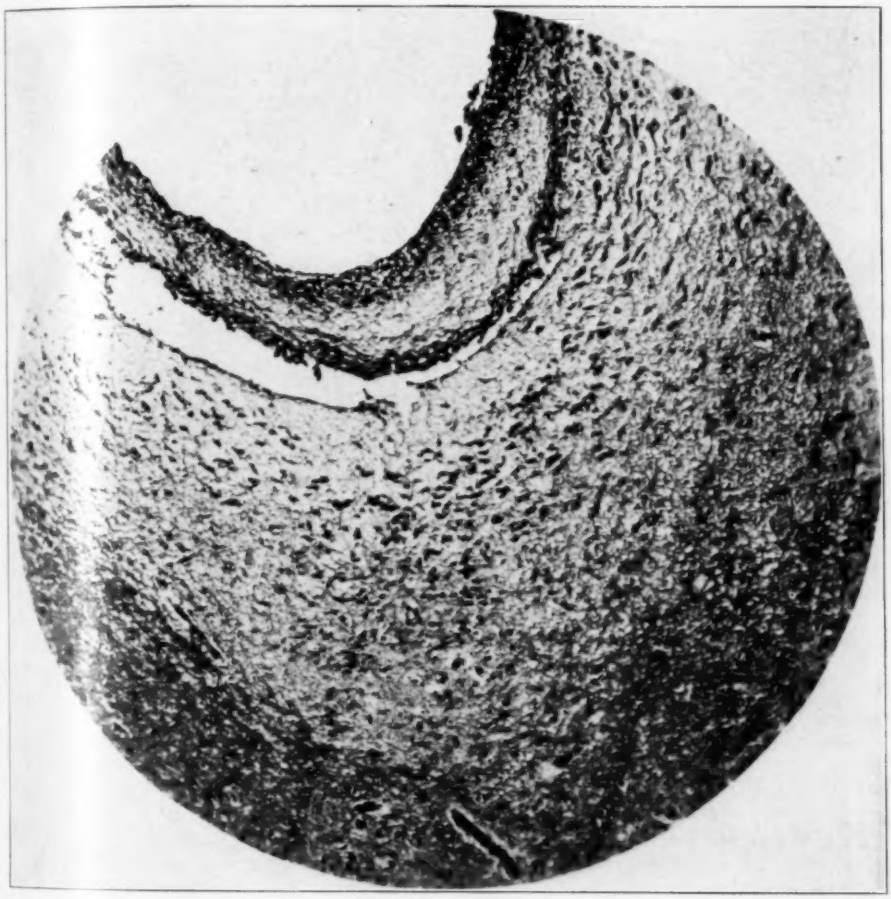


Fig. 5.—Section of the aqueduct of Sylvius, complete loss of the ependymal lining and its replacement by a band of hyalinized tissue. The subependyma shows a wide zone of gliosis.

beyond this and the numerous corpora amylacea pathologic changes were not noted. It is significant that careful search failed to disclose any evidence of inflammatory reaction in the choroid plexus.

Summary and Comment.—The anatomic observations indicate the presence of an inflammatory process limited mainly to the ependymal lining of the entire ventricular system of the brain. The mild inflam-

matory and degenerative changes seen in the adjacent brain tissue were little more than a reaction to a contiguous inflammatory process. They certainly do not warrant consideration as part of the picture of the disease, and do not justify an assumption that this is an encephalitis or encephalo-ependymitic process. In the widespread ependymitis there were present all gradations, from the mildly acute purulent in the lateral

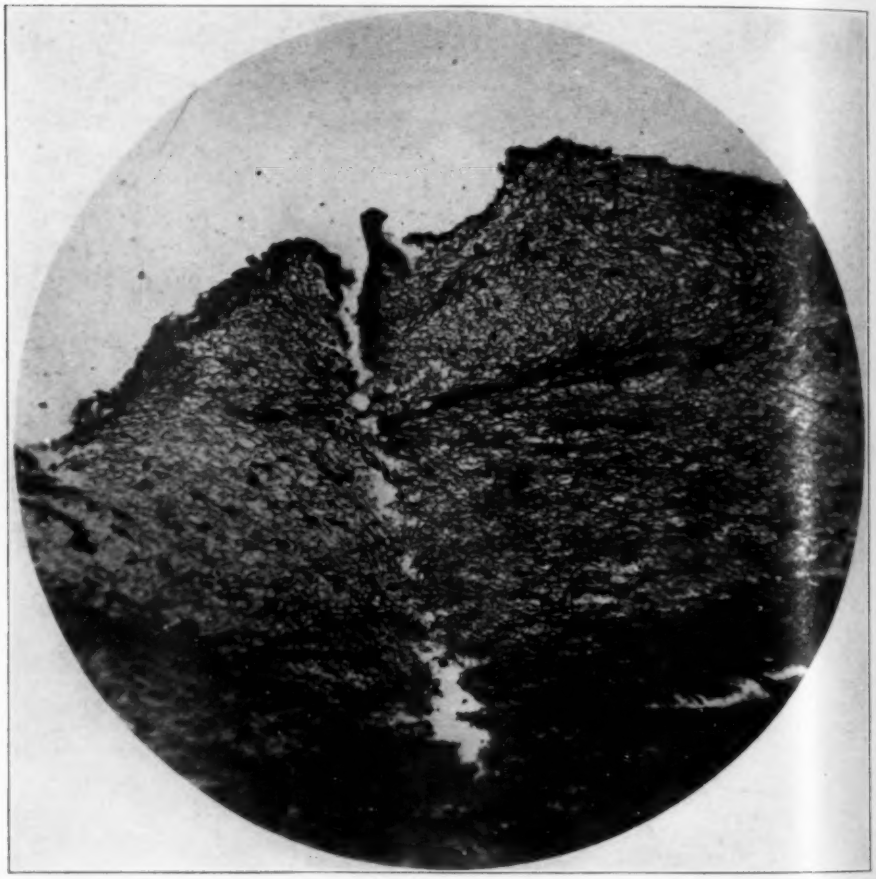


Fig. 6.—Aqueduct of Sylvius with a typical ependymal granulation.

ventricle to the chronic granular ependymitis in the aqueduct and the fourth ventricle. However, a subacute inflammation predominated. This strongly suggests that at least some instances of chronic granular ependymitis have their beginning in mild and slowly progressing acute forms.

While, as has already been pointed out by others, ependymitis is quite often encountered both in the acute and in the chronic form, the subacute type of primary ependymitis, as in our case, is somewhat

rare. In the literature, there are only a few examples (Mérle,[5] Tillgren,[6] Spiller,[7] Cushing[9]); among them, the case of Tillgren is of particular interest, for it presents anatomic observations and clinical features strikingly similar to those in our case.

Tillgren's Case:[6] In his case, that of a man, aged 25, a rather sudden onset of illness was followed by the gradual development of manifestations of increased

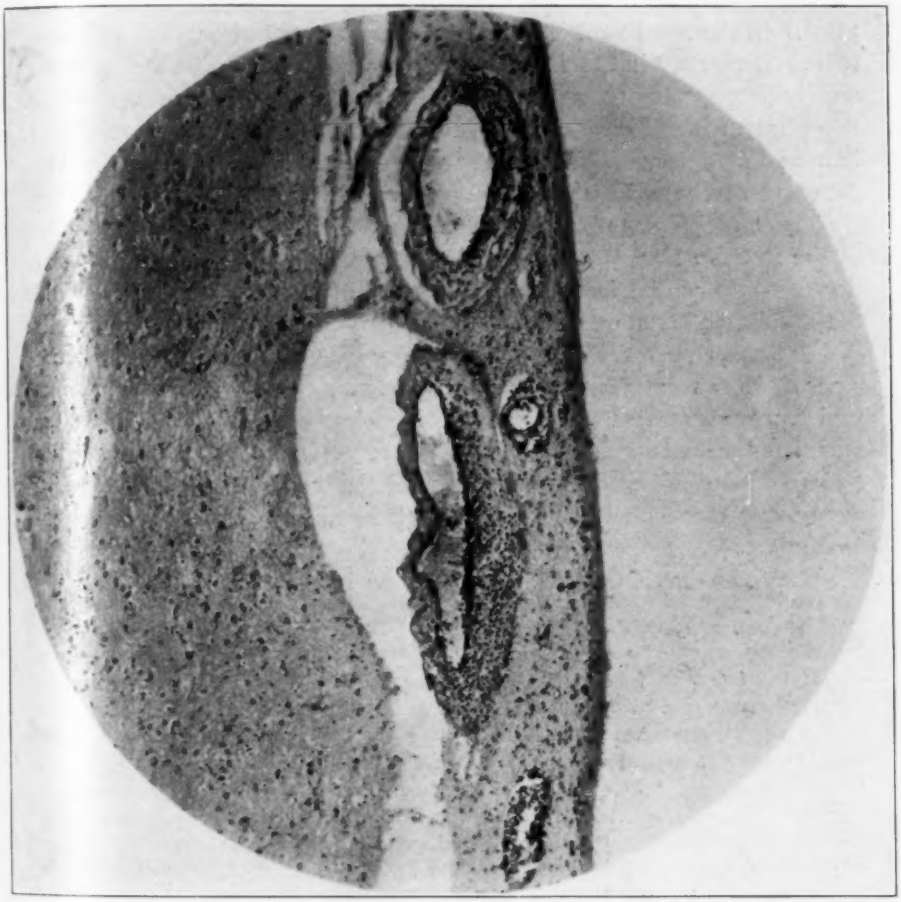


Fig. 7.—Section of the wall of the pseudocyst.

tension. Early in the clinical course there were periods of dimness of vision, attacks of vomiting, generalized muscular weakness and coarse muscular twitchings. Short remissions of relative well-being were followed by short aphasic episodes associated with cortical discharges to the right side of the face and, later, occasional attacks with loss of consciousness. The objective neurologic observations were limited to slight weakness of the right angle of the mouth, exaggerated knee jerks and bilateral ankle clonus. Shortly before death, the patient passed through a more profound jacksonian attack, during which he became aphasic; the eyes were turned to the left, the left side of the face was in tonic contraction, the

left arm was in tonic spasm, soon followed by clonic spasm in the right arm. The head became retracted. The patient passed into stupor and died one hour after the onset of this attack.

Besides the jacksonian attacks, there was a striking clinical parallelism to our case; still more striking, however, was the similarity in the anatomic observations. Grossly, the brain showed evidence of increased intracranial tension. All ventricles were markedly dilated, especially the third. In the third ventricle the ependyma was thickened, and this was still more marked in the fourth ventricle, where it assumed a gray color and had a gelatinous appearance causing marked narrowing of the lateral recesses. The thickened lining of the fourth ventricle extended into a pointed, tonguelike structure which closed the central canal. The aqueduct of Sylvius was reduced to a small lumen.

The most pronounced histologic changes were found in the lining of the fourth ventricle. The latter was completely replaced by granular tissue, which varied locally in the density of its connective tissue and the cellular infiltration. Where the fibrous tissue was dense, the cells of infiltration were few; here, hyalinizing changes were noted. In the areas of loose connective tissue there were many cells of a polymorphous variety; among these were many polynuclear giant cells. The latter stimulated an unsuccessful search for tubercle bacilli as well as for the *Spirochacta pallida*. In other parts of the ventricular system, there were many ependymal granulations with herniating masses of proliferated glia cells.

The following is Cushing's description of his case [8]:

A patient, some months after an acute attack of so-called "brain fever," presented symptoms that were supposed to be those of tumor—profound headache of varying intensity, located in the left side; choked disk, more pronounced in the left eye; attacks of vomiting, aphasia, and weakness of the body musculature of the right side. A simple decompressive operation was performed, unfortunately without aspiration of the ventricle. The patient's symptoms were not appreciably altered, and some weeks later he died from bulbar paralysis due to pressure. At autopsy a dilatation of the left ventricle alone was found. The choroid plexus appeared hypertrophic and greatly enlarged. The ependyma was studded with sago-like granulations and the left foramen of Monro was occluded. The ventricular linings elsewhere were perfectly normal in appearance and there was no trace of thickening of the basal meninges suggestive of an earlier meningeal infection.

In commenting on the case, Cushing says that: "The process, doubtless, often bears some relation to preexisting meningeal disturbances, whether infective or not. Thus, chronic acquired hydrocephalus of the adult, giving intermittent symptoms and showing granular ependymal thickenings, may represent the chronic stage of serous meningitis of the ventricular form." And, again, that "Primary ependymitis with ventricular hydrops may closely resemble those cases of tumor which, early in their course and before there are localizing symptoms, lead to

obstructive hydrocephalus. Both conditions result in bilateral spastic paralysis if the lesion is symmetrical. Occasionally there is a unilateral dilatation of one ventricle. A fluctuation of symptoms is more probable in ependymitis than in hydrocephalus from tumor. A cytological examination of the lumbar fluid may help."

GENERAL COMMENT AND CONCLUSIONS

Uncommon anatomic observations in the nature of subacute diffuse ependymitis are here described in a case which manifested clinical signs and symptoms of cerebral neoplasm. The productive inflammatory changes in the ependymal lining, with scar formation, led to closure of the foramina of Monro and of the aqueduct of Sylvius, in this way completely sealing the third ventricle anteriorly and posteriorly. The ependymal lining of this ventricle was also involved in the process, was thickened and gave rise to a structure which assumed the character of a cyst. This condition, added to the internal hydrocephalus which resulted from the closure of the foramina of Monro, was responsible for the bilaterality of neurologic signs and for the cardinal symptoms of tumor cerebri.

The clinical picture, in the absence of true neoplastic growth as verified by observations at autopsy would justify the grouping of our case with the so-called pseudotumor cerebri, were it not for the fact that this term is now disqualified by recent contributors on the subject. It is agreed that, since in a large number of instances, one or more of the so-called cardinal signs and symptoms of tumor of the brain may be provoked by a variety of nonneoplastic pathologic changes in the brain, it would be misleading to assemble them under the vague, anatomically meaningless term, "pseudotumor cerebri."

The clinical manifestations in our case, as in the few recorded similar cases, cannot be regarded as pathognomonic of the provoking disease process. Though the subacute and chronic forms of ependymitis are fairly well established as isolated and primary conditions of the central nervous system and constitute a pathologic entity, they do not yield signs or symptoms that enable one to recognize these conditions of disease during the life of the patient. However, the occurrence of remissions in a case of chronic internal hydrocephalus should, as pointed out by Cushing, put one on guard for such a pathologic process.

The presence of several transitional stages in the inflammatory ependymal process in one and the same case is of equal importance; in our case acute, subacute and chronic phases were represented. This feature supports the belief that many of the so-called chronic forms of ependymitis, including granular ependymitis, especially when they are associated with an inflammatory process elsewhere in the central nervous system, as in general paralysis, are the results in the end of a slowly progressive form of inflammation of the lining of the ventricular system.

RABIES IN MAN

MICROSCOPIC OBSERVATIONS *

KONSTANTIN LOWENBERG, M.D.
Pathologist, Foxborough State Hospital
FOXBOROUGH, MASS.

Modern methods of prevention have met with such success in most civilized countries that little opportunity presents itself for further study of rabies either clinically or pathologically. Within six months, at the psychiatric clinic of the University of Minsk, Russia, six patients infected with this virus died. Postmortem examinations were performed in four of these cases, and a microscopic examination of the lesions was made with special consideration of the problems presented in the recent work of Schukri and Spatz.[1] Starting from the point of view presented in a previous work by Klarfeld,[2] these authors attempted to draw a parallel between the histologic picture in rabies and that in epidemic encephalitis. They found that "in both cases of rabies investigated, the inflammatory changes were of a type and localization quite similar to those found by numerous observers in cases of encephalitis epidemica." The two cases differed from one another in that in the second case a marked degenerative process was added to the inflammatory one. In both cases, however, the inflammatory phenomena were "most strikingly localized in the substantia nigra, that center which seems also to bear the brunt of the attack in encephalitis epidemica." The authors concluded that the similarity of the histologic picture of the two processes was so great that "without knowledge of the history and clinical course of the disease, one would undoubtedly, from the histologic picture alone, consider first the diagnosis of encephalitis epidemica." Of especial interest in their work is the consideration of a possible relation between the length of the period of incubation and the type of histologic picture found. In the following work, I shall reconsider these problems on the basis of a thorough analysis of the microscopic observations made on my material. An excellent summary of previous work on the subject is given by Schukri and Spatz, and I will restrict discussion of the literature to their work and that of Klarfeld, especially since their investigations have brought to light an altogether different aspect of the problem.

My material consists of the following four cases:

---

* From the neurological division of the University of Minsk, White Russia.

1. Schukri, I., and Spatz, Hugo: Ztschr. f. d. ges. Neurol. u. Psychiat. **97**: 627, 1925.

2. Klarfeld, B.: Ztschr. f. d. ges. Neurol. u. Psychiat. **77**:148, 1922.

REPORT OF CASES

CASE 1.—A. K., a Russian peasant boy, aged 15, was bitten and scratched on the right hand by a rabid cat in October, 1925 (the cat was afterwards killed by three dogs, one of which was in turn killed by a wolf some four weeks later, whereas the other two are alive and do not show signs of the disease). The patient did not receive prophylactic treatment. About the middle of March, 1926, he began to feel weak and generally indisposed (the presence of fever could not be ascertained). A few days later, he suddenly developed deglutition spasms and marked restlessness. He was admitted to the psychiatric division of the second city hospital in Minsk, on March 15, 1926, when moribund, and he died six hours later. I performed an autopsy eight hours after death.

Postmortem Observations.—The skull and the dura were normal. The leptomeninges were soft but of a slightly reddish tinge in places. The convolutions appeared grossly normal. Frontal sections through the brain showed that the cortex was of normal width and color and distinctly outlined. The white matter was apparently normal. The lateral ventricles were not dilated. The basal ganglia were well outlined. The substantia nigra was almost unrecognizable. The ependyma and basal vessels were soft. The cord, as well as all other organs, did not show any gross pathologic lesions.

Histologic Examination.—The pia did not show anything of significance with the exception of occasional fresh hemorrhages and rare lymphocytes in the adventitia. With the exception of a few rarefactions, the greater part of the architecture of the cortex was well preserved. The ganglion cells, however, were stained rather homogeneously in a grayish-blue tint. The tigroid substance could not be distinguished. The protoplasm showed marked shrinking. Outside of the nucleus, which was fairly well preserved with a well staining nucleolus, the rest of the components of the cell body were hardly recognizable. In some cases, the whole protoplasm was converted into a dustlike mass without signs of structural relations but with a preserved nucleus. Some cells took on the form of grayish-blue netlike structures that had lost all normal outline, while others were reduced to cell shadows. The cortical neuroglia was generally unaffected, except for a slight swelling of the scavenger cells and the occasional occurrence of Hortega cells. The blood vessels were not particularly affected, showing only a slight swelling of the endothelia and rare lymphocytes in the adventitia. A somewhat more marked tendency toward degeneration was found in the cortex of the cornu ammonis: poorly staining cells, disappearance of the tigroid substance and shrinking down to complete destruction of the whole cell. Inflammatory processes were not found in the cortex.

In the midbrain, the cellular element of the substantia nigra was the part most heavily affected. The cystoplasm was reduced to debris, the nucleus scarcely recognizable, the pigment practically destroyed, leaving small greenish particles on the borders of the cell body. The whole field was filled with numerous cell remains and shadows. The thick swollen cell processes stained deeply, intertwining to form a thick network. The neuroglia cells were increased in number, their nuclei markedly swollen, and there were numerous protoplasmatic elements. Large neuroglial nodules replaced the nigra formation at numerous points. All these neuroglial reaction processes with numerous neuronophages controlling the picture seemed to be more or less restricted to the sites of destroyed parenchyma (fig. 1). The moderate infiltrations about the vessels accentuated the inflammatory character of these phenomena. Quite similar in kind, if not in degree, were the destructive processes in the other components of the midbrain. Here, too, they took the form of diffuse neuroglial proliferation, especially rich in

protoplasmic elements, the disappearing nerve cells becoming centers of active neuronophagia. The inflammatory character of the process predominated here also.

A similar process, if not as intense, was met with in the thalamus and striate body. Here the infiltration, as well as the neuroglial proliferation, was more moderate; neuronophagic processes were not noted but the parenchyma was markedly affected. In the spinal cord, from the dorsal segments downward, the process was mostly degenerative with tigrolysis and loss of parenchymatous tissue, but without inflammatory phenomena. The cervical segments, however, as well as the medulla, showed a moderate but distinct inflammatory process. The Bielschowsky-stained sections merely confirmed the observations in those cell processes stained by the Nissl method. The fibrous neuroglia did not show proliferation, but the sections stained with sudan III showed occasional fat drops in the cortex and midbrain.

Case 2.—A. T., a Russian peasant, aged 35, was bitten on both hands by a rabid dog in February, 1926. Three days after infection, prophylactic treatment

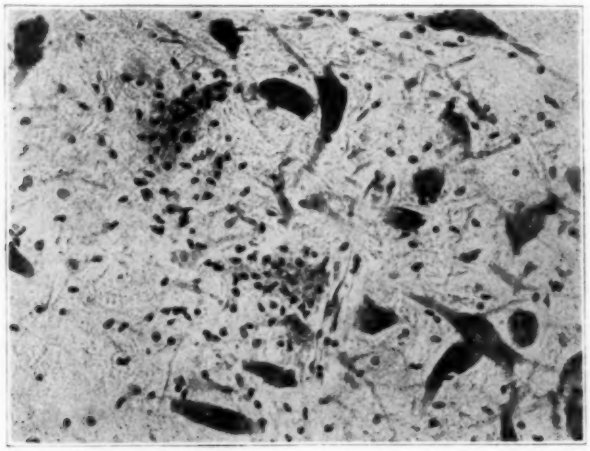


Fig. 1.—Substantia nigra, showing distinct neuronophagies; marked degeneration of the pigment bearing cells with heavily staining processes; cell shadows, and diffuse neuroglia proliferation. Nissl stain; × 200.

(Hogyes) was begun. The extent of the treatment was not ascertained. The onset of the disease occurred in July, 1926, with general weakness and slight fever, followed on the third day by marked restlessness, acute deglutition spasms and difficulty in swallowing. He was admitted to the same hospital on July 7. On admission, he showed beginning paresis and some deglutition spasms, but the sensorium was clear. The patient had insight into the seriousness of the disease and told the physician that he knew he was going to die, but that he would not infect anybody else. He submitted to lumbar puncture, after which he grew worse. He retained clear consciousness although muttering to himself, and begged for water. He died forty-eight hours after admission. Autopsy was performed four hours after death.

Postmortem Observations.—The skull and dura were normal. The leptomeninges were somewhat hyperemic. The frontal sections through the cerebrum showed that the cortex was grossly normal. There were numerous small hemorrhages in the white matter. The basal ganglia appeared to be of normal outline without apparent foci. On the floor of the fourth ventricle, there were a few

small fresh hemorrhages. The ependyma and the vessels of the base were soft. Pathologic observations were not made in the other organs.

Microscopic Examination.—The pia showed numerous fresh hemorrhages, but otherwise was unchanged. In the cortex, the changes in the nerve cells were similar to those in the first case. In places there were slight neuroglial proliferations and an increase in size and number of the scavenger cells, but there were no infiltrations, and the vascular system was apparently normal.

The substantia nigra was uniformly affected to a marked degree. The nerve cells, in part altogether destroyed with some pigmented substance left behind and in part markedly degenerated, resembled those seen in case 1. The dark greenish pigment left behind was in process of being taken up by gitter cells and protoplasmic neuroglia. This pigment seemed to preserve its normal characteristics for a long time after the disappearance of the mother cell, and was found in the neuroglia or lying free in the surrounding tissues. As in the first case, there were numerous neuronophagic processes. The neuroglial reaction was further expressed in the formation of thick nodules as well as in diffuse proliferations throughout the substantia nigra. There were occasional Hortega cells, small lymphocytic infiltrations and free leukocytes in the tissues. Almost all vessels showed lymphocytic infiltrations. The neuroglial cells and the rest of the tissue substances showed marked edematous swelling. In the pons and surrounding tissues, the changes resembled those found in the first case, namely, neuroglial nodules, neuronophagic processes and lymphocytic infiltrations in the blood vessels and lying freely in the tissue.

Basal Ganglia: The changes in the thalamus and striate body took the form of extensive degenerations of the parenchyma, slight neuroglial proliferation and formation of occasional neuroglial nodules. Infiltrations into the vessels or in other areas and neuronophages could not be found. In the thalamus were small masses of peculiar greenish pigment which at times were free in the surrounding tissues, and at other times were taken up by the neuroglial elements. There were from 4 to 6 of these foci in each field. Bielschowsky, sudan III and victoria blue stains did not reveal anything of significance. In the white matter, there was a moderate nuclear proliferation.

Case 3.—I. M., a Russian peasant, aged 32, was bitten on the chest and both hands by a rabid dog on May 12, 1926. Antirabic prophylaxis was begun the same day, but was interrupted by the patient after the seventh injection. Four days later, he came back for further treatment. The patient had to walk 50 kilometers daily to reach the hospital. On June 14, the patient was admitted to the district hospital with a typical picture of rabies, with restlessness and deglutition spasms. Two days later, he was being transferred to the Minsk Psychiatric Clinic, but died on the way. Autopsy was performed three hours after death.

Postmortem Observations.—The skull and dura were normal. The leptomeninges had a deep bloody tinge. Frontal sections showed that the cortex was of normal width, but bluish in color. The white matter was studded with numerous small hemorrhages. Grossly, there were no focal lesions. The basal ganglia were well outlined. On the floor of the fourth ventricle were numerous small fresh hemorrhages in the medulla as well as in the pons. The other organs did not reveal any pathologic changes.

Histologic Examination.—The pia was markedly edematous and contained many gitter cells, lymphocytes and macrophages, as well as occasional leukocytes in its network. There were also some fresh hemorrhages. The endothelial cells

of the vessel walls were slightly swollen. The disturbances in the cortex were similar to those noted in the previous cases, but were not as extensive. There were no infiltration phenomena, the vessels being intact. Slight degenerations of the parenchyma cells were seen in the cornu ammoni.

The substantia nigra showed a neuroglial proliferation with numerous nodules of different sizes. The parenchyma was markedly affected. There was shrinking of the pyramidal cells with disappearance of the nuclei, the whole cell having been reduced to a black, shapeless structure; the number of cells was reduced. The pigment-bearing cells were practically all destroyed. Neuronophagic processes could not be found. The process in the pons and surrounding structures in this case was similar to that in the substantia nigra, the parenchyma of the gray nuclei showing degenerative changes varying in degree, and at times, an almost complete destruction of the elements. Here, however, there were numerous neuronophagic processes and a marked neuroglial proliferation. The neuroglial reaction was concentrated about the parenchyma, whereas the white matter remained practically untouched, showing slight proliferations about the blood

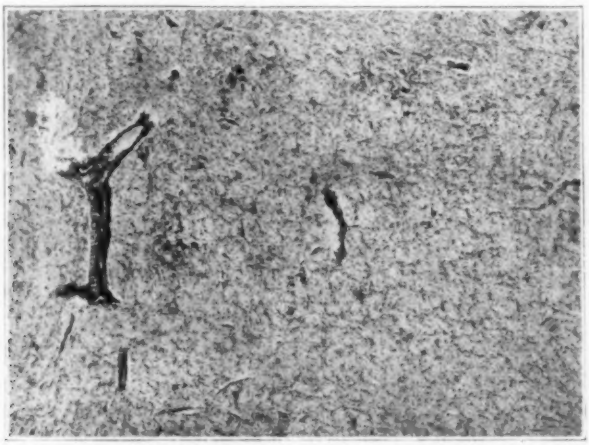


Fig. 2.—Substantia grisea centralis, showing typical picture of an intense inflammatory process; marked lymphocytic infiltration of blood vessel and lymphocytic and leukocytic invasion into the tissue; marked neuroglia proliferation and degeneration of the parenchyma. Nissl stain; × 40.

vessels only. The blood vessels throughout these regions showed lymphocytic infiltrations. The whole picture was that of an acute inflammatory process. In the thalamus and striate body there was a preponderance of degenerative disturbances with only slight inflammatory phenomena; the cells were markedly affected, the neuroglia was slightly proliferated, and there were occasional infiltrations into the vessels. As in the previous case, the Bielschowsky, sudan III and Weigert stains did not bring out any new points.

Case 4.—K. L., a Russian peasant girl, aged 5, was bitten on the left corner of the mouth by a rabid dog on Oct. 12, 1926. On October 15, prophylactic treatment was instituted, but on October 25 (while the treatment was still being given), the first symptoms of the disease appeared. On October 27, she was admitted to the hospital presenting a typical picture, with restlessness and deglutition spasms. Lumbar puncture on the same day showed: fluid, turbid; high pressure; a slightly positive Nonne reaction and negative Pandy and Weichbrodt

tests; cells 1,032, mostly leukocytes; paraffin and mastic tests were positive in the second and third tubes; colloidal gold test, 0343210000. The patient died on October 28 and autopsy was performed three hours after death.

Postmortem Observations.—Superficially, one could see the scars at the site where the patient was bitten. The skull was normal. The dura was taut. The vessels of the leptomeninges were markedly injected; the leptomeninges themselves, however, were normal. The surface of the brain appeared dry and had a bluish-red tinge. The convolutions were flattened. Frontal sections through the cerebrum showed the cortex to be of normal width, with grayish-blue tinge; there were scattered fresh hemorrhages. The vessels were dilated and filled with blood. The white matter showed a similar richness of blood vessels. The basal ganglia were similar in appearance to the cortex. On the floor of the fourth ventricle, there was a fresh hemorrhage about the size of a small pea. A similar one was found in the substantia nigra. Further foci were not found. The cerebellar cortex was grossly like that of the cerebrum. The meninges of the spinal cord were markedly injected.

Histologic Examination.—The leptomeninges contained a great many leukocytes and lymphocytes. The vessels were dilated, and there were numerous small fresh hemorrhages. The cortex showed an extensive, purely degenerative disturbance, as in cases 1 and 2. The vessels showed slight infiltrations, some of the cells being leukocytes. Here and there were small fresh hemorrhages, apparently brought about by necrosis of the vessels (this will be discussed later). The basal ganglia showed a purely degenerative process. The neuroglia retained a rather passive attitude both in the basal ganglia and in the cortex. The white matter showed a slight, nuclear proliferation.

Inflammatory phenomena were found in the pons (mostly in the gray nuclei), with the exception of the stratum profundum pontis, in the whole substantia nigra, and especially in the centralis grisea, where they reached the highest point (fig. 2). The red nucleus showed the inflammatory phenomena to a lesser degree, and the medulla, spinal cord and cerebellum showed a purely degenerative process. The inflammatory phenomena in the substantia nigra, pons and red nucleus were, generally speaking, quite similar to those found in case 3, but there was a preponderance of leukocytic infiltration here and absence of neuronophagic processes. The histologic changes in the vessels of the substantia nigra and especially those of the substantia grisea centralis showed marked deviations from the changes found in the first three cases (fig. 3). The vascular changes were of two types: 1. A smaller number showed swelling of the wall elements (mostly adventitial), so much so that the lumen was at times obliterated. In vessels of this type, there were either no infiltrations or moderate lymphocytic ones. 2. A type of change was frequently seen in the substantia grisea centralis; in this type, the infiltrations controlled the picture and consisted almost exclusively of leukocytes. The adventitia was literally filled with them, and the whole vessel was surrounded by a broad band of cells, a picture which closely resembled that of a purulent inflammation. There was more or less marked necrosis of the adventitial network and of the leukocytes themselves. The different elements of this structure showed different stages of necrotic destruction; in some places this necrotic process was restricted to the adventitia, but in others it also broke through into the lumen, causing the small hemorrhages previously noted throughout the brain (fig. 4).

The occurrence of leukocytes was not restricted to the adventitia. They worked their way into the brain substance and were found there in masses of different shapes or scattered throughout the tissue, lending the whole picture the aspect of purulent inflammation. The energetic neuroglial reaction to the inflam-

matory process helped to emphasize the acuteness of the lesion. The necrotic process was restricted to the vascular system, however, the nerve tissue being spared. Occasionally one found abscess-like leukocytic nodules, which on further examination proved to be vascular infiltrations; the vessel (frequently a capillary) was either altogether covered by the infiltration or had been destroyed in the necrosis so that only a few small remains were left behind. Pure abscesses were not found anywhere in the tissue.

COMMENT

A review of the microscopic observations in these cases brings out the following characteristic facts: As far as the inflammatory phenomena were concerned, the brunt of attack seemed to be borne by the substantia nigra and the substantia centralis grisea and, to a lesser degree, by the pontile nuclei. In all of these, the reactive phenomena were strikingly localized about the parenchyma. The proliferation of the neuroglia seemed to be restricted to the region of the function-bearing tissue.

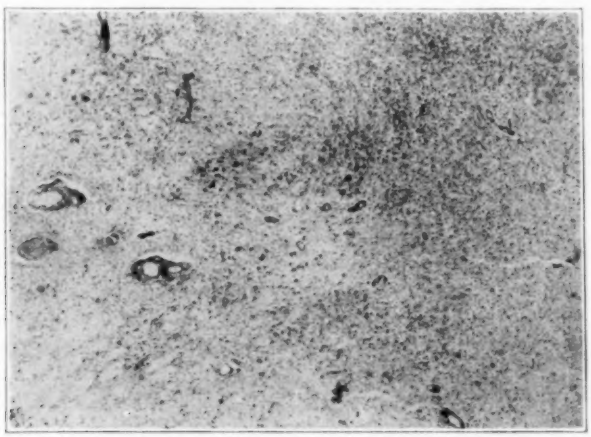


Fig. 3.—Substantia grisea centralis, showing marked leukocytic infiltrations into the vessel wall and deeper into the surrounding tissues. Nissl stain; × 40.

This was especially well seen in the substantia nigra. The pigmented elements of this center showed the most distinct neuronophagia, and where the cells were completely destroyed there was marked phagocytosis of the pigment by the neuroglia. A similar restriction of neuroglial proliferation to the sphere of influence of ganglion cells that were undergoing destruction was seen in the pontile nuclei, whereas in the white matter there seemed to be almost no signs of inflammatory changes.

The type of neuroglial cells most frequently found were special types of Hortega cells with large, well staining nuclei either having narrow strips of protoplasm or being altogether devoid of it. Occasionally, one found the rodlike form of Hortega cells and, infrequently, large protoplasmic elements were found. All these neuroglial cells were, as a rule, scattered throughout the affected tissues, with occasional

Babes nodule formations and other attempts at organized neuroglial structure. Underlying this picture there seemed to be a generalized edema of the substratum. The vascular elements were also affected but not extensively, and in these a lymphocytic infiltration seemed to control the picture. This fundamental picture was especially characteristic of cases 1 and 2, whereas cases 3 and 4 showed some deviations. In case 3 were first encountered signs of an acute meningitis: leukocytes were seen in the affected regions, and there was a much more energetic infiltration about the blood vessels. The substantia grisea centralis also, which was markedly affected in all cases, was affected more in case 3, and particularly strikingly in case 4, in which it seemed to be the central point (as far as intensity goes) of the whole process. The character of the inflammatory process seemed to gain new features. The vessels were markedly affected; the infiltration was purely leukocytic and quite as

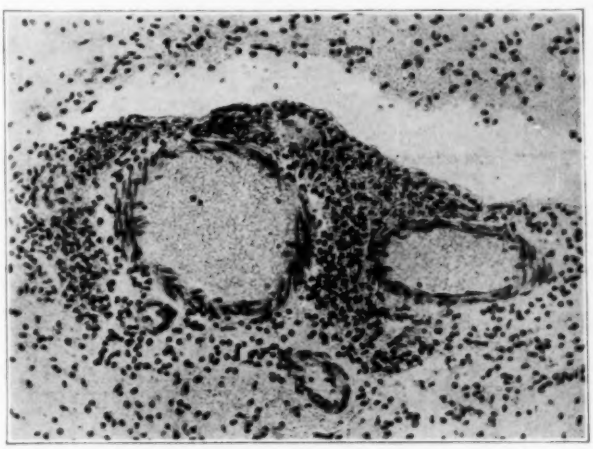


Fig. 4.—Substantia grisea centralis, showing purely leukocytic infiltration about the blood vessels and the invasion into the surrounding tissues. Nissl stain; × 200.

intensive in the smallest capillaries as in the larger vessels. In this case, the infiltrations seemed to reach abscess-like proportions, the similarity with a pus-forming process being further intensified by the exudation of leukocytes into the parenchyma. The parenchyma itself did not show any tendency toward necrosis, but occasionally there were necrotic foci in the vessel walls which lent the picture an aspect not unlike that of encephalitis purulenta. The meningitic component added to the picture was not altogether the same in these two cases. In case 3, the condition was not an acute meningitis, for there were occasional gitter cells, macrophages, a few lymphocytes, marked edema and occasional hemorrhages, in contrast to which the picture in case 4 was that of an acute purulent meningitis. The changes in the cortex were predominantly those of a degenerative type, inflammatory phenomena being rare and found mostly about the vessels.

The following microscopic picture can be established as characteristic of the central nervous system lesions in rabies: (1) inflammatory processes localized in the midbrain, and especially marked in the substantia nigra (Schukri and Spatz),[1] substantia grisea centralis and red nucleus; occasionally also in the meninges; (2) a predominantly degenerative lesion in the cortex, basal ganglia, white matter and cerebellum. Thus far my observations are in principle the same as those of Klarfeld and Schukri and Spatz, who came to the conclusion that the microscopic changes in rabies are much like those in epidemic encephalitis. My first two cases undoubtedly seem to point in that direction. In case 3, however, and especially in case 4, there are changes which, even if possible, are yet extremely unusual in epidemic encephalitis. The meningitic component in case 3 and the purulent inflammatory process and its localization in case 4 seem to lend the picture a characteristic aspect.

As a further point of interest, I would like to emphasize that the character of the cerebrospinal fluid is unlike that in other acute disease processes. The observations in case 4, although showing a pleocytosis and therefore in keeping with the acute meningitic process, do not show any of the other meningitic reactions (Nonne, Pandy, colloidal gold tests, etc.). In the cerebrospinal fluid examinations in the other cases there seemed to be the same lack of organic contents; furthermore, a normal cell count in those cases does not show any meningitic involvement.

The clinical course and the microscopic observations in the four cases reported would seem to bear out the hypothesis advanced by Schukri and Spatz that the incubation period bears a definite relation to the intensity of the pathologic process. Case 4, in which the incubation period was nineteen days, showed the most marked inflammatory process, whereas in the first two cases, with incubation periods of over six months, there was no meningitic involvement and the degenerative process was more distinct.

SUMMARY

1. A microscopic study of the nerve lesions in four cases of human rabies is reported and the observations are discussed in relation to those of other investigators.

2. Both inflammatory and degenerative lesions were found, with a tendency of the former to localize in the midbrain (substantia nigra and substantia grisea) and at times also in the meninges; degenerative lesions had a tendency to localize in the cortex and basal ganglia.

3. The observations in two of the cases showed a similarity to those of epidemic encephalitis. In the other two, however, new elements were added to the picture, tending to give it a character of its own.

4. There appears to be a relation between the length of the period of incubation and the character of the lesion. The shorter this period, the more acute and intense is the process.

THE MEASUREMENT OF CEREBRAL AND CEREBELLAR SURFACES

IV. A TECHNIC FOR MAKING ENDOCRANIAL CASTS SUITABLE FOR THE ESTIMATION OF THE INTERNAL SURFACE OF THE SKULL OVERLYING THE CORTEX CEREBRI *

WALTER M. KRAUS, M.D.
NEW YORK

Investigation of the area of the surface of endocranial casts (fig. 1) overlying the cortex cerebri of modern and primitive man was begun at the suggestion of Dr. Frederick Tilney, who lent me the casts. Dr. William K. Gregory of the American Museum of Natural History also

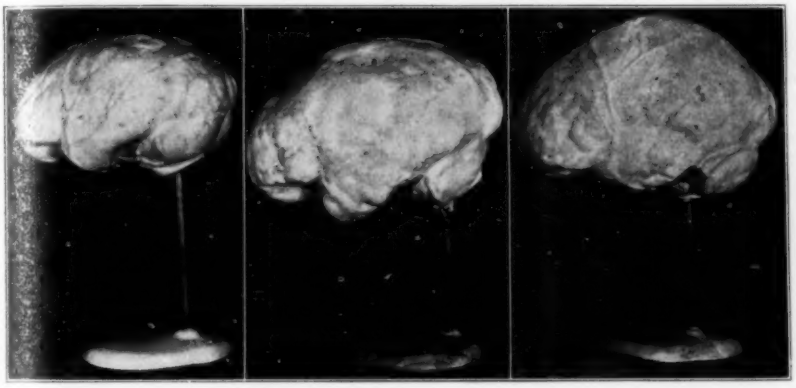


Fig. 1.—The original endocranial casts made of plaster.

lent me a cast of a normal skull. Mr. Bell of the museum made the piece molds of these casts.

The problem of making a cast from these molds which could be precisely cut into slabs was difficult. Various mixtures of wax and paraffin were inappropriate. A knife would not cut them, and the mixture was too sticky to use with an electrically run band-saw. The saw was continually pulled off the wheel on which it ran.

After many attempts with various mixtures, the following formula and method was obtained which fulfilled all requirements:

---

* From the Neuropathology Laboratory, Montefiore Hospital.

* This work was carried out under the auspices of the Hilda Stich Stroock Fellowship.

* Read before the Fifty-Third Annual Meeting of the American Neurological Association, Atlantic City, N. J., May, 1927.

1. Mix and melt:

Cornstarch	1 part
Beeswax	1 part
Solid paraffin, melting point F. 68-72 C	3 parts
Rosin	1 part

Slowly pour this mixture after heating and melting it into the piece-mold after this has first been soaked in water to allow the air to leave the plaster.

2. Do not open the piece-mold until the following day; then remove the cast and put it in a wooden box.

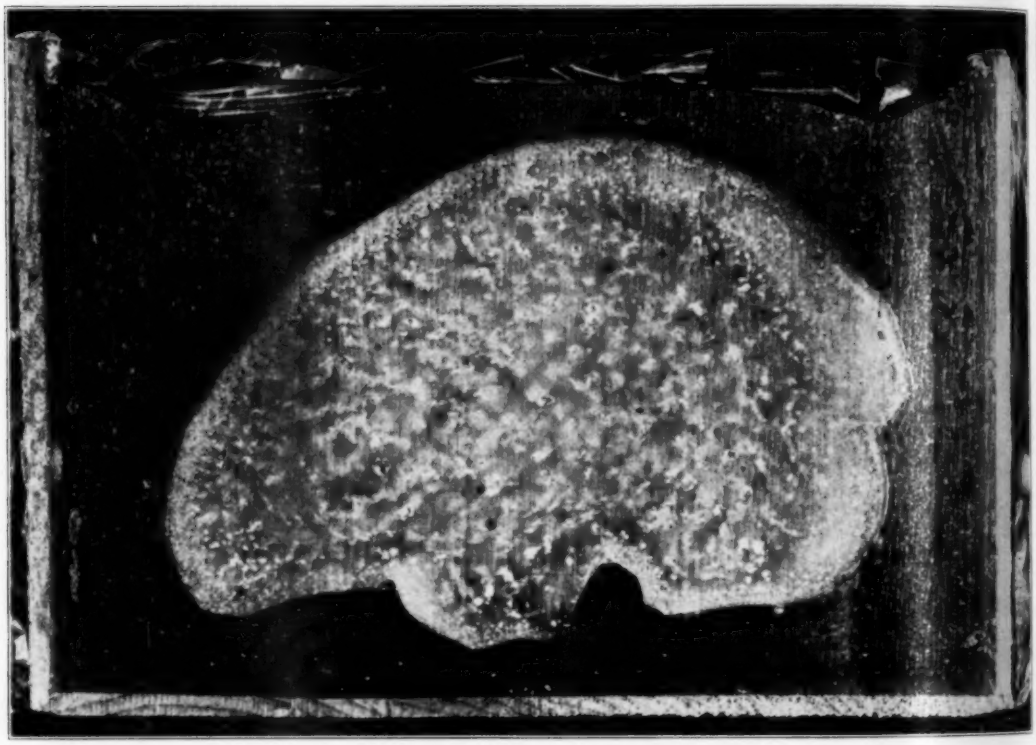


Fig. 2.—A slab cut from one of the casts made by the new formula.

3. Fill the box with the following warm mixture until the cast is completely covered:

Paraffin	3 parts
Rosin	1 part
Sudan III (a small amount) dissolved in 100 cc. of xylol	

This mixture has to be used as cool as possible, just a little over the melting point.

4. When this base is hard and cool, put the box in an icebox over night and cut the following morning with a band-saw in slices of 1 cm. each. Oiled

tissue paper should be placed between each section to prevent them from sticking together. Figure 2 shows one of these cut slabs.

Before cutting, remove all nails from the box in order to save the saw.

5. These sections can now be measured according to the method given by Kraus and Ditto,[1] and the results used to determine the internal surface of the skull overlying the cortex cerebri.

---

1. Kraus and Ditto: A Method of Measuring the Cerebral and Cerebellar Cortical Surfaces, Arch. Neurol. & Psychiat. **17**:193 (Feb.) 1927.

ATYPICAL NEURALGIA

UNSUCCESSFUL ATTEMPTS TO RELIEVE PATIENTS BY OPERATIONS ON THE CERVICAL SYMPATHETIC SYSTEM *

CHARLES H. FRAZIER, M.D.
PHILADELPHIA

In the neurosurgical clinic of the University Hospital, major trigeminal neuralgia and atypical forms of neuralgia, because of the wealth of material, have occupied a considerable amount of the time of the attending staff. The files contain records of 1,287 cases, of which 1,083 were classified as typical (major trigeminal neuralgia) and 204 as atypical. Altogether, the major operation has been performed 458 times. I have felt it more or less incumbent to issue a statement as to the nature of these atypical forms of neuralgia (a paper on this subject is in process of preparation) and to publish experiences with operations on the cervical sympathetic system for relief from pain.

Sympathectomies of one kind or another might be said to be the last venture in surgery as a means of relieving pain, though years ago Jonnesco proposed removal of the cervical sympathetic ganglion for relief in trigeminal neuralgia. It has always been a disappointment to me that the operation failed, since, had it succeeded, one might have had a clue as to the origin of this baffling disease, perhaps on the basis of an arterial ischemia of vasomotor origin. The trigeminal tract has a liberal sympathetic connection through its otic, its ciliary and its sphenopalatine ganglion, not to speak of the relation of the gasserian ganglion itself with the sympathetic plexus of the internal carotid artery. I recall one patient who, fifteen years after a sensory root operation, showed a typical Horner's syndrome. Though there is a fairly intimate knowledge of these various sympathetic anastomoses with that great sensory nerve, the trigeminus, it has served little or no purpose.

Leriche's operation, periarterial sympathectomy, has been extraordinarily effective in relieving pain in certain instances; but as yet there has not been a satisfactory explanation for this, if it is true, as generally accepted, that the sensory fibers supplying the blood vessels for the most part run centrally in the spinal nerves and not for any considerable distance in the periarterial sympathetic plexuses.[1]

The third outstanding example of operations on the sympathetic system for relief from pain is that for relief in angina pectoris. The literature of 1924, 1925 and 1926, abounded with communications from

* Read at a Meeting of the Philadelphia Neurological Society, April 22, 1927

1. Wiedhopf: München. med. Wchnschr. **72**:417, 1925.

laboratory and clinic, discussing this welcome suggestion, but the end-results have been disappointing in many instances. Just why is not yet apparent, since the operation is founded on reasonably substantial anatomic facts. Edgworth [2] and Ranson [3] have shown a direct connection between the middle and inferior cervical sympathetic ganglia and large sensory fibers in the cardiac plexus from the vagus and upper three thoracic nerves.

I might include in this category of pain-freeing procedures by operations on the sympathetic system, the operation for the relief of gastric crisis or section of the splanchnic nerve for the relief of gastric pain of other origin,[4] but that would be going too far afield.

What is known of the anatomy of the sympathetic system that is pertinent to this thesis? In the first place, there is a distinction between the cervical and the thoracic or lumbosacral ganglia. The cervical sympathetic trunk is composed exclusively of preganglionic efferent fibers, which are derived from the upper thoracic nerves through the white rami and terminate in the cervical sympathetic ganglia. Ranson,[5] contrary to a statement which I have seen made, has demonstrated that the cervical sympathetic ganglia do not, like the other ganglia, have any gray rami or postganglionic fibers that arise in the ganglion and relay the impulses onward. The efferent preganglionic fibers arise from the upper thoracic spinal nerves as well as from the third, seventh, ninth and tenth cranial nerves.

The superior cervical ganglion is said to send communicating branches to the first and second cervical nerves, to the petrous ganglion of the glossopharyngeal nerve, to the hypoglossal nerve, to the pharynx, a branch to the carotid plexus, which in turn communicates with the gasserian ganglion and the great deep petrosal nerve, and to the cavernous sinus.

Stimulation of the cervical sympathetic system has been known to cause widening of the pupil, widening of the palpebral fissure, protrusion of the eyeball, lacrimation, vasoconstriction of the vessels of the face, unilateral sweating and changes in the salivary secretion, and Karplus has demonstrated a pilomotor reaction. These phenomena are of irritative origin, and the trophic disturbances that follow paralysis of the cervical sympathetics are everyday observances.

If sympathetic fibers of the cervical chain, however, have sensory representations in the region of the face, why is it that electric stimulation, which causes those other phenomena, does not cause pain? In

2. Edgworth, F. H.: J. Physiol. **13**:260, 1892.
3. Ranson: J. Comp. Neurol. **29**:313, 1918.
4. Braun: Zentralbl. f. Chir. **48**:1038, 1921.
5. Ranson: Sympathetic Nervous System, J. A. M. A. **86**:1887 (June 19) 1926.

the appended clinical records are recorded a number of observations made on conscious patients. In a number of operations performed under local anesthesia, before resection of the ganglia or stripping of the arterial plexus, these structures were stimulated with the electrode, but in only one instance (case 9) was there a suggestion or an intimation from the patient, of pain in any part of the face. Pain was complained of at the site of stimulation but nowhere else. These observations, repeated as they have been, seem to me to carry great significance and cannot be disregarded.

Müller, in his monograph (Die Lebensnerven), says that it is doubtful whether the cervical sympathetic nerves serve as carriers of sensory impulses. It is not known whether irritation of this structure causes pain or paralysis or any evidence of sensory disturbances. In this respect, the cervical sympathetic nerves differ from the sympathetic fibers of the abdomen, as represented in the splanchnic system, the sensory function of which can be easily demonstrated.

In 1924, Heuer [6] noted in a patient, who had been operated on eight years before consulting him and had not been completely relieved, that pressure on the superior cervical ganglion caused severe pain similar to the original attacks, although the entire trigeminal zone was anesthetic. This observation has been repeated by Fay and others in my own clinic, but always in a group of patients who are extremely susceptible to suggestion and whose nervous thresholds are low. In one instance, the patient complained of tenderness, only on the side opposite that in which the pain picture occurred. I have observed that the application of ligatures to the superior thyroid artery in the conscious patient has caused pain in the trigeminal distribution, an observation confirmed by others; but to this particular instance of pain syndrome, in the absence of other confirmatory evidence, little importance can be attached.

Perhaps the most significant evidence, if it can be confirmed, as to the existence of sensory function in the cervical sympathetic system has been supplied by the examination of patients whose cervical sympathetic ganglia have been removed for relief in angina pectoris. It has been observed frequently that patients, while relieved of the anginoid pains, have complained of pain in the trigeminal and occipital regions and in the shoulder and arm. A few surgeons,[7] however, have reported certain sensory disturbances following sympathectomy for angina pectoris. Perhaps the most striking of these observations was made by

6. Heuer, quoted by Reid: Sensory Disturbances Following Sympathectomy for Angina Pectoris, J. A. M. A. **83**:113 (July 12) 1924.

7. Bacon, J. H.: Left Superior Cervical Sympathectomy Under Local Anesthesia in Angina Pectoris, J. A. M. A. **81**:2112 (Dec. 22) 1923: Reid (footnote 6).

Reid, who found in the trigeminal zone, as well as elsewhere, that the epicritic sensibility was absent, protopathic sensibility diminished, superficial pain reduced, and the threshold for pressure pain markedly lowered, with disturbances of temperature sense and sense of position. He stated that the function of nearly all the cranial nerves that have an intimate association with the superior cervical ganglion, except the fourth and sixth, was disturbed. So frequently has excision of the superior cervical ganglion been practiced (over 200 times by Jonnesco alone) that it is surprising these striking phenomena have not been noted before. In my own clinic, a number of examinations have been made (Dr. E. C. Russell) to determine whether after section of the sensory root and later resection of the cervical sympathetic ganglion there has been any change in the pressure sense. Up to the time this paper was written the observations have been entirely negative.

Reid [7] says: "It has been the experience of almost every surgeon who has had a large experience that division of the sensory root has failed to relieve the symptoms thought to be due to the disease." This is, as I believe, only another way of saying that those particular surgeons had made a mistake in diagnosis, and it does not follow, as Reid suggests, if the evidence which I am about to present is accepted, that the sympathetic system may be more at fault than the trigeminal nerve.

Because of the suspicion that has been aroused as to the part the cervical sympathetic system plays as an etiologic factor in these varied pain phenomena, mostly in the trigeminal distribution, it seemed eminently proper that a series of cases should be put on record in which the superior cervical ganglion had been removed and the periarterial sympathetic plexus stripped. In my service at the University Hospital, this operation has been performed thirteen times by several operators (Grant, Muller, Frazier). From this series, I have selected ten cases for presentation. For the sake of brevity I present only in abstract the essential data. Of this series, the sensory root operation in five instances had been performed, and in all but one of these no doubt there had been an error in diagnosis. The patients probably did not have true trigeminal neuralgia.

REPORT OF CASES

CASE 1.—A woman, aged 56, was admitted to the neurosurgical clinic on March 22, 1916. She was operated on March 23, (section of the sensory root). Some features of the neuralgia were atypical, but others seemed sufficiently characteristic to justify the major operation.

Course.—March 7, 1917: Following the operation, the patient complained bitterly of a burning, throbbing sensation in the upper and lower aveolar processes and in the malar region. These disturbing sensations continued night and day

without intervals of relief, and on one occasion it was noted that there was a vasomotor flush on the affected side when the pain was severe.

The patient was seen a number of times in the course of the seven intervening years until May 6, 1923, when she was readmitted to the University Hospital. On May 2, the superior cervical sympathetic ganglion was excised and the periarterial sympathetic plexus stripped from the common carotid artery from a point 2.5 cm. below the bifurcation to 2 cm. above the external carotid artery.

Result.—April 11, 1927: the patient wrote that she was worse rather than better, and that she was never without a pulling, burning or throbbing sensation in the cheek. She did not refer to the gums, which before the operation were also the seat of discomfort.

Case 2.—A woman, aged 53, was admitted to the neurosurgical service on Jan. 20, 1923. She had certain atypical features like the patient in the first case. The radical operation had been performed, and during the year following the patient developed a psychosis and was under treatment for eight months.

June 6, 1924: On her second admission, the patient stated that the pain was worse than before the operation; she described it as "drawing" and "pulling," and referred it to the whole trigeminal distribution.

June 10: Dr. Grant stripped the periarterial plexus of the common cartoid artery for a distance of 5 cm. and excised the superior and middle ganglia and intermediate trunk.

December 3: Six months after the operation, the patient continued to complain of a drawing, pulling pain, and stated that she was worse rather than better.

Case 3.—A woman, aged 58, was admitted to the neurosurgical service on Nov. 12, 1921. The description of the pain corresponded closely to that of major trigeminal neuralgia. There seemed to be no atypical features.

November 18: The radical operation was performed, the motor root being conserved. Convalescence was uneventful.

April 12, 1923: On her second admission, it was found that the patient had developed certain paresthesias.

April 12: Dr. Muller stripped the periarterial plexus for a distance of 6 cm. and excised the superior cervical sympathetic ganglion.

Feb. 28, 1927: The patient said that she was entirely relieved for nine months, when the disturbing sensation returned. Now it is described as "drawing sensations" in the face, beginning at the ala of the nose and extending to the vertex.

Case 4.—A woman, aged, 56, was referred to the Neurosurgical Service on May 4, 1925, with a diagnosis of major trigeminal neuralgia. There was considerable doubt as to the diagnosis and a deep alcoholic injection of the mandibular division was given without relief.

March 6, 1926: On her second admission, she continued to complain as before. In the interval, because of a positive Wassermann reaction, she had received intensive treatment in the Syphilis Clinic without avail. There were certain features of her discomforts which suggested the possibility of a tumor of the gasserian ganglion. The region of the ganglion was explored. No tumor was found; the ganglion was excised.

Feb. 7, 1927: On her third admission, the patient described the pain as "boring" and "burning," "hot boiling," and referred it to the left side of the face, including the left side of the tongue and alveolar processes. Recently pain had been experienced in the right mandible.

February 18: Dr. Grant removed the superior cervical sympathetic ganglion on both right and left sides, and stripped the periarterial sympathetic plexus on

both sides. It was noted at the operation that electrical stimulation of the ganglia and the periarterial plexuses did not excite pain in any part of the face.

Result.—April 12: The patient reported the pain just as before; it was worse rather than better.

CASE 5.—A woman, aged 40, was admitted to the Neurosurgical Service on April 14, 1924, with the following complaint: The first symptom developed six years before admission when she was awakened one night with a severe pain at the outer, lower portion of the right malar bone. The next morning she was able to open the mouth only slightly. Extraction of teeth and medical treatment failed to give relief; nine months later, an operation in the right temporal region was performed at the Brigham Hospital in Boston. Following this operation she was able to open the mouth wider than before, and the pain, which had been constant and much worse at night, while still constant was severe only for an hour or two each day. About one year later, she had a severe burning in the throat, which lasted six weeks and disappeared spontaneously. In January, 1923, the pain became more severe than ever, and in July and August of that year, she received several injections of alcohol, all inside the mouth which did not give any relief. In September, a second operation in the temporal region was performed at the Brigham Hospital, and in November, in Providence, a third operation was performed through the same scar.

When the pain recurred after the third operation it was described as a constant, severe boring pain deep in the malar region; a severe burning sensation under the eye; burning sensation in the throat since the operation in September; sensation of pricking of needles in the right side of the forehead; aching in the entire right lower jaw. There was never any cessation of pain, and every few days there was an excruciating, lightning-like pain in the malar region, lasting but a second. Both lips were involved on the right side, and occasionally the right side of the tongue felt as though a knife were being run through it. A sensation as of dust in the eye was constant, but there was no pain in the lids. Occasional pain in the right ear and occasional ache in the left side of the face were present, but the later was never severe. Eating, chewing, talking, swallowing, drinking cold water and touching the face aggravated the pain. The patient had taken large quantities of morphine without relief. She never slept more than an hour each night.

A complete physical and neurologic examination revealed nothing except anesthesia in the entire trigeminal distribution, with enophthalmos and narrowing of the palpebral fissure on the side on which the operations had been performed before admission to the University Hospital. There was said to be an intense pain on pressure over the right carotid artery, but none over the left.

May 1: Dr. Muller excised the middle and superior cervical sympathetic ganglia with the intervening trunk and stripped 4 cm. of the periarterial network. The upper and lower segments were injected with 95 per cent alcohol.

Result.—The patient died in August, 1926, but she had never experienced any relief.

CASE 6.—A woman, aged 47, was admitted to the neurosurgical service on April 26, 1924, and described her discomforts as follows: Four years before presentation she began to have rather severe pain in the left temporal region. The pain radiated gradually over the left frontal and malar regions. It gradually became worse. At the same time she began to experience boring pain in the

left occipital region. She believed that the occipital pain was more troublesome than the other, since it was constant and she always had a spot of tenderness in that region. At the end of six months, ⅛ grain (0.0081 Gm.) of morphine was required to relieve the pain. The dose was increased, as the pain increased, to ⅙ and ¼ grain (0.010 and 0.016 Gm.) during the second year. During the year before admission to the hospital, three doses of morphine, ½ grain (0.0324 Gm.) were necessary to give relief. The pain was sharp and shooting in the left frontal, temporal and malar regions and sometimes settled as a constant, severe neuralgic pain, which persisted for a day if not relieved. The pain in the occipital region was not sharp, but rather a steady, boring ache. It never passed the midline of the head, but sometimes shot down to the midline of the chin and midline of the forehead about the left eye, back behind the left ear and down the neck, then out to the point of the left shoulder. Occasionally, the pain was in the left side of the neck behind and below the angle of the jaw. The pain now came on suddenly as a sharp stabbing pain, which then settled down to a constant ache. The outer surface of the left upper alveolus sometimes was affected. The patient never had had any facial tic or muscular spasm. She became nauseated during an attack and occasionally vomited. The attacks recurred every five or six weeks at first, then became more frequent until she had them every two or three days. For the last few months before admission, they had been coming every two or three weeks and were precipitated by fatigue, excitement, cold, emotional stress or jolting.

A thorough physical and neurologic examination did not elicit anything of significance. A careful analysis of the painful sensations suggested an irregular form of migraine, with certain tender points, especially at the angle of the jaw, behind the mastoid process and in the supra-orbital region. Together with the severe and mild headaches, there were, as described, certain shooting pains, a pallor about the eye and puffiness of the eyelids.

May 12, 1924: The superior cervical sympathetic ganglion with about 6 cm. of the trunk was excised; the periarterial plexus was stripped, and the peripheral and central segments were injected with 95 per cent alcohol.

Result.—June 23, 1925: A letter from the attending physician, Dr. Edward King, stated that she was suffering as before the operation.

Case 7.—A woman, aged 40, admitted to the neurosurgical service on June 13, 1924, complained that about May 5, 1923, the sensory root had been sectioned for relief in a case of major trigeminal neuralgia. This gave entire relief from the pain. The patient regained weight, but subsequently began to complain of a sense of pressure in the temporal region, as though there was something in the tissues which should not be there, and of pressure and a drawing sensation in the left eye, as though it were too large for the socket. These discomforts were worse at night.

Physical and neurologic examinations gave negative results, except for an area of anesthesia in the trigeminal zone.

Operation.—June 25, 1924: The superficial cervical sympathetic ganglion and 2 cm. of the trunk were excised, and 3 cm. of the periarterial plexus, 3 cm. below the bifurcation was stripped. It was noted that electric stimulation: (1) of the common carotid artery caused no referred pain; (2) of the superior thyroid artery excited pain up the neck to the face; (3) of the cervical sympathetic trunk caused a sense of vibration in the left eye and head, and (4) of the sympathetic ganglion elicited pain in and about the left eye.

Result.—Five days after the operation, the pressure sensation had disappeared. Three years later, on April 11, 1927, the patient wrote that she was "no better";

whereas, before the operation, there was more of a pulling and drawing sensation of the eye, now there was a "continual burning sensation as though the eye were under an awful strain."

CASE 8.—A woman, aged 33, was admitted to the neurosurgical clinic on June 18, 1924, with the complaint of severe attacks of pain of thirteen years' duration, coming on usually at the menstrual period and lasting two or three days. The pain began at the root of the nose, extended to the temporal region, in front of the ear, behind the ear and in the infra-orbital region. The attacks were accompanied by vomiting and were aggravated by fatigue or excitement. Drooping of the lid, injection of the conjunctiva and lacrimation were associated with the attacks. The pain was described as "dull, grinding, right into the bone." The patient had been accustomed to taking hypodermics during the attacks.

A physical and neurologic examination did not reveal anything of significance. A subtotal hysterectomy had been performed in 1919.

Operation.—June 27, 1924: The superior cervical sympathetic ganglion was excised and the periarterial plexus was stripped. Stimulation of the common carotid artery with an electrode caused pain in the lower jaw.

Result.—The patient was seen on Jan. 9, 1925, and said that she had not had relief. The pain is now worse than before the operation, especially beneath the infra-orbital ridge and just above the supra-orbital foramen. The pain which she had in front and behind the ear has disappeared. Recently, there has been pain on the opposite side of the face.

CASE 9.—A woman, aged 32, admitted to the neurosurgical clinic of the University Hospital on Sept. 8, 1924, complained that seven years before, following the birth of her second child, she had her first attack of pain. She described it as "throbbing" and "pulling," and it was referred to the malar and temporal regions. The attacks at first were synchronous with the menstrual period, but later were experienced in the intervals. They lasted about twenty-four hours, and were associated with vomiting but not with vasomotor symptoms. During the attack, she said she could not move the eyeball.

A physical and neurosurgical examination did not elicit evidence of an organic lesion. The function of the cranial nerves was not disturbed.

Operation.—Sept. 24, 1924: Under local anesthesia, the superior cervical sympathetic ganglion was removed; the trunk was crushed before its entrance at the base of the skull. The periarterial plexus was stripped for a distance of 2 cm. and a ligature was applied to the common carotid artery above and below the stripped area. Stimulation with the electrode: (1) of the common periarterial plexus after it had been isolated but not divided did not cause pain; (2) of the common carotid artery before the plexus was isolated caused pain referred to the temple, upper teeth and side of the nose; (3) of the cervical sympathetic trunk did not cause pain.

Result.—In March, 1925, seven months after the operation, the patient wrote that she was still suffering agony. On April 11, 1927, she reported that she was not relieved of the discomforts; she still had pain in the temple, eye and face, and when she yawned there was cramp in her neck.

CASE 10.—A man, aged 46, was admitted to the neurosurgical clinic of the University Hospital on March 4, 1920, with every evidence of major trigeminal neuralgia. The radical operation was performed, with complete relief from the neuralgic symptoms. Subsequently, he began to complain of a sense of pins and needles in the face, a stinging sensation in the gums and cheeks, a ringing in the ear

and a sense of dryness in the surface of the eye. This was regarded as a form of paresthesia, possibly of sympathetic origin, and he was readmitted to the hospital for a second operation on March 13, 1923.

Operation.—The periarterial plexus of the common carotid artery was stripped for a distance of 4 cm. Stimulation of the artery with an electrode before stripping caused (1) a sense of pressure in the gums and (2) a sensation like an electric shock in the left side of the neck. Stimulation of the external carotid artery caused a sense of pain in the upper jaw and forehead. After stripping, stimulation of the common carotid plexus did not elicit a painful sensation, and stimulation of the external carotid plexus caused a feeling of pressure in the wound.

COMMENT

The records speak for themselves. In only one case did the patient obtain any relief, and in this case, there still remains the doubt as to whether the relief was permanent as the patient cannot be traced. In one instance, the patient said that she was relieved for only nine months.

These observations, if accepted, demonstrate rather conclusively that if patients continue to have discomfort after the major operation for trigeminal neuralgia, an operation on the cervical sympathetic ganglion does not offer any hope of relief. Furthermore, the operation is equally ineffective in that group of painful syndromes which, for want of a better term, I have been in the habit of calling "atypical" neuralgias. It will be noted in the cases reported, as in the majority of the cases in this atypical group, that the predominant discomfort is described by the patient as a boring, throbbing, burning pain. Frequently, there is a sense of deep pressure. One or two of the patients exhibited in the rather complicated picture (I say complicated advisedly, for often it is extremely difficult to elicit from the patient a concise and constant, unvarying description of the discomforts) symptoms suggestive of migraine. But no matter what the kind and character of the pain, in most instances, the operation not only did not afford relief but even aggravated the condition. The patient invariably complained of being worse rather than better; this bears out what I believe to be true of all operative undertakings (of which maxillary sinus drainage is the most common) in these atypical cases. Any attempt by physical measures to afford relief not only fails in its original intent, but aggravates rather than improves the condition.

The question arises: Is this experience with these ten selected cases, in which the superior and sometimes the middle cervical ganglion was removed and the periarterial plexus stripped, convincing? Is one justified from this experience in stating baldly that (whether the sympathetic system carries sensory fibers or not) the sympathetic system has nothing to do with these peculiar pains? Can one go a step further and say there is no such thing as sympathetic neuralgia? These are

queries that should soon be answered. Recently, there has been much loose talk about the part the sympathetic nerve plays in atypical neuralgia; it is high time that this hypothesis should be either proved or disproved. Only a few days ago, a patient reported to me that a well known authority in neurologic problems had told her that her pain syndrome, which was unquestionably not tic douloureux, was a form of "sympathetic" neuralgia.

Is there the slightest confirmatory evidence for this hypothesis? If there is, how can it be proved? I am prepared, on the evidence presented by the material of this contribution, to dismiss as untenable the sympathetic theory at once. The critic may not be satisfied with my demonstration on the grounds that the operation was in most instances confined to the superior ganglion and the adjacent trunk. Surely there would be nothing to gain by including the middle and inferior ganglia in the resection. Do the branches from these ganglia preside over any sensory function in the region of the face? I think not. If, as I once thought, these painful zones correspond to vascular zones, zones corresponding to the distribution of given branches of the external carotid artery, resection of the periarterial plexus should have a beneficent effect, but it has not. One would expect, if this postulate were true, that there would be at least a period of transitory relief and would explain the return of pain on the grounds of regeneration. But in the ten cases there was only one, possibly two patients who were relieved by operation. In one of these the relief was only transitory; in the remainder the patient not only was not better, but was worse.

This problem is of some importance because the number of patients afflicted with conditions that have these irregular form pictures is not small, and the patients themselves lead a wretched existence. That one of the patients was entirely relieved by Christian science suggests a functional rather than an organic basis for the pain, and one may have to be content to view the condition as of psychogenic origin and discontinue any effort to modify or relieve it by physical procedures. This would be the easiest course to pursue. If one is not content with this, what is the next step in an investigative inquiry as to the true nature of this pain picture?

ELECTROMYOGRAPHIC STUDIES IN DIFFERENT TYPES OF NEUROMUSCULAR DISTURBANCES *

CURT P. RICHTER, PH.D.
AND
FRANK R. FORD, M.D.
BALTIMORE

Knowledge of what occurs in the nerves and muscles during a voluntary muscular contraction is still meager, since it is not known even to what extent such a contraction is dependent on reflex mechanisms.[1] Extensive work has been done on isolated neuromuscular preparations, but from such experiments alone one cannot hope to understand the activity in the nerves and muscles of the intact body. The chief method available at present for this purpose is that of recording the action currents which may be led off from the contracting muscle, and it is probable that this will prove to be the most satisfactory procedure. The only other method tried thus far, that of recording the muscle sounds, seems to be filled with so many uncontrolled factors that it is practically useless.

Gasser and Newcomer,[2] in records taken simultaneously from the efferent nerve and from the contracting muscle itself (phrenic nerve and diaphragm), showed that the waves of the electromyogram correspond exactly with the nerve impulses arriving at the muscle from the central nervous system. From the action currents set up by the muscles, therefore, one can learn what goes on in the nervous system. This knowledge should be especially useful in the examination of persons with nervous lesions of such type that normal muscular activity is no longer possible. A study of pathologic conditions, however, presupposes an understanding of the electromyogram from normal persons, and there is still some difference of opinion regarding the nature of normal action currents. The regular rhythm of 50 per second reported by Piper [3] from voluntary contractions, for instance, has not been generally confirmed, since such records are found consistently in only a few persons. Further difficulty has arisen from the failure of most workers to distinguish the different components of the electromyogram. Normal voluntary action currents appear to consist of at least three different parts: (1) the primary waves; (2) the Nebenzacken, and (3) the secondary waves.

* From the Johns Hopkins Medical School.

1. An excellent summary of our present knowledge of the electromyogram from reflex and voluntary contractions is given in Fulton's Muscular Contraction and the Reflex Control of Movement, Williams & Wilkins Company, Baltimore, 1926.

2. Gasser and Newcomer: Am. J. Physiol. **57**:1, 1921.

3. Piper: Electrophysiologie menschlicher Muskeln, Berlin, 1912.

The primary waves often show great irregularity in shape and amplitude, and their frequency varies from 5 to 100 per second, increasing with the tension applied to the muscle (Wachholder,[4] Richter [5]), and decreasing with fatigue (Piper,[3] Cobb and Forbes [6]). Moreover, the frequency varies with the muscle involved, the currents rising slowly in some muscles and rapidly in others. Weizsäcker [7] established the theory, however, that these large waves are not specific for voluntary contractions, as was formerly supposed, when he demonstrated their presence in the electromyogram from reflex contractions of the leg muscles in cases in which all voluntary movements of the legs were eliminated by a complete transection of the cord. Richter showed recently that the large waves were prominent in the electromyogram from the grasping reflex of the new-born infant, and the evidence seemed to be conclusive that their origin was somewhere within the reflex arc, either in the motor horn cells or in the proprioceptive end-organs in the contracting muscle. He found also that the frequency of these waves increased with the tension applied to the muscle and decreased with fatigue, according to principles similar to those developed by Adrian and Zottermann [8] for the proprioceptive impulses. Weizsäcker contended that, in addition to the impulses from the proprioceptive end-organs, the electromyogram was affected by impulses from the anterior horn cells, and he postulated for these cells an inherent rhythm of a constant frequency near 15 per second. Dusser de Barenne and Brevée [9] also believed that an inherent rhythm of the anterior horn cells was expressed in the electromyogram, but they found that the rate was between 50 and 60 per second, rather than 15, as reported by Weizsäcker.

The Nebenzacken are small projections which appear to be superimposed on the primary waves. They are irregular in both amplitude and frequency, and their presence depends on the rate of the large waves. When the primary waves occur at a frequency of about 40 per second, the Nebenzacken are conspicuous, but then they gradually disappear until, at a rate of 60 per second in the muscles of the forearm, and 75 per second in the biceps, they are completely absent. Their presence seems to indicate that the proprioceptive impulses are out of phase.

The secondary waves have a small amplitude, which seems to be controlled largely by the tension of the galvanometer string, since they usually appear with a tight string, distributed with comparative regularity throughout the entire record. When they can be counted, their

4. Wachholder: Arch. f. d. ges. Physiol. **199**:595, 1923.
5. Richter: Quart. J. Physiol. **18**:55, 1927.
6. Cobb and Forbes: Am. J. Physiol. **65**:234, 1923.
7. Weizsäcker: Deutsche Ztschr. f. Nervenh. **70**:115, 1921.
8. Adrian and Zottermann: J. Physiol. **61**:151, 1926.
9. Dusser de Barenne: Skandin. Arch. f. Physiol. **43**:107, 1923. Dusser de Barenne and Brevée: J. Physiol. **61**:81, 1926.

rate varies from 125 to 250 per second, and they seem to depend to a large extent on impulses from higher centers.

Most workers have found tha the electromyogram in pathologic conditions is similar to that obtained from healthy muscles. There are, however, a few exceptions, some of the most important appearing in observations made by Weizsäcker on two patients with "néurotabes périphérique" of Dejerine. In these patients the frequency of the large waves was reduced from the normal average of 50 to 60 per second to 10 to 15 per second, and at the same time the amplitude seemed greater than normal. Gregor and Schilder [10] and Weizsäcker have found that in severe cases of tabes, the frequency of the large waves is diminished, but that in milder cases the electromyogram is normal. In a case of dystrophia myotonica Adrian [11] also found regular waves, but the amplitude was greater than in any of our cases of muscular dystrophy.

We have taken electromyographic records in a large number of cases representing almost all types of neuromuscular disorders. For a long time, in agreement with previous observations, we found no distinct differences in the records. Then we examined a case of syringomyelia in which the records deviated widely from the normal in that the frequency of the primary waves was strikingly reduced and the amplitude of each individual wave was increased. Similar currents were obtained later in a patient with progressive muscular atrophy. An attempt was made, therefore, to determine the significance of these abnormalities in the electromyogram. Pathologically, what had these two cases in common? We found that in each condition the anterior horn cells were affected. Accordingly, we studied the electromyograms from four cases of poliomyelitis, in which, again, it was known that the anterior horn cells were involved. The records from all four of these patients showed the peculiarities discovered in the curves from the cases of syringomyelia and of progressive muscular atrophy.

Since the muscles from which the records were taken were markedly atrophied in every case, it was possible that the reduction in rate might be due to the atrophied condition rather than to the lesions of the anterior horn cells. Records were taken, therefore, of patients with muscular dystrophy in which a marked atrophy of the muscles was produced by a disease process localized in the muscles and not involving the nervous system. All these patients showed abnormal currents different from those of the patients with lesions of the anterior horn cells.

In order to determine further whether the peculiar records obtained in the cases of anterior horn cell lesions were due simply to the interruption of the reflex arc, or specifically to the lesions of the anterior horn cells, records were taken in several cases of tabes. Here, in agree-

10. Gregor and Schilder: Ztschr. f. d. ges. Neurol. u. Psychiat. **14**:359, 1913.
11. Adrian: Lancet **1**:1229 and 1282, 1925.

ment with the previous observations,[12] we obtained records essentially normal, except in advanced cases in which the large waves seemed to disappear altogether leaving only the small waves.

In the present paper these different records will be described in detail and discussed on the basis of our present theories regarding the origin of the various components of the electromyogram.

METHODS

Essentially the same methods were used as were described in previous papers.[13] The currents were recorded by means of an Einthoven string galvanometer (Hindle model). The electrodes consisted of small pieces of pure zinc sheeting, about ¾ inch (1.9 cm.) square, covered with a thin layer of paste made by mixing kaolin and saturated zinc sulphate solution. In order that monophasic currents might be obtained, one electrode was attached over the belly of the muscle to be studied and the other to the shell of one ear. A puncture was made with a hypodermic needle through the skin above the muscle and behind the ear before the electrodes were applied, in order to eliminate the resistance of the skin, which in some persons is high. It is possible, then, to work with the same conditions in each subject, and with a fairly tight string at all times. Most of the records were taken on either the biceps or the muscles of the forearm, since more is known about the electromyogram from these muscles and more satisfactory records are obtained from them than from other muscles. In each patient records were taken usually of voluntary extension and flexion with and without resistance, and also of passive flexion and extension.

RESULTS

CASE 1.—*Syringomyelia. Cavity involving the anterior horn cells in segments C_7 and C_8, the spinothalamic tracts from segments C_8 to T_2, the pyramidal tracts and the right nucleus ambiguus.*

Rosa H., aged 38, had suffered from weakness and numbness of the arms and had had difficulty in swallowing for several years. Examination disclosed that the right side of the soft palate and of the pharynx was weak and that the right vocal cord did not move. The deltoids, spinati, biceps, brachialis anticus and extensors of the wrists were markedly atrophied and extremely weak, the weakness being greater on the right than on the left side. The legs were strong, but slightly spastic. Loss of cutaneous sensibility for pain and temperature was found over both arms and from the chin to the second rib in front and from the occipital protuberance to the level of the spinous process of the third thoracic vertebra behind. There were scars of unfelt burns over the shoulders and hands. Tactile sensibility was slightly diminished over the same area. The biceps and supinator jerks were not obtained; the triceps, knee and ankle jerks were increased. There was a bilateral Hoffman hand reflex and a bilateral extensor response on plantar stimulation. The Wassermann reactions on the blood and spinal fluid were negative.

Electromyogram.—Records of the action currents were taken from the biceps and the muscles of the forearm on both sides. The biceps on the right side was

12. Gregor and Schilder (footnote 10) and Weizsäcker (footnote 7).

13. Richter: New Methods of Obtaining Electromyogram and Electrocardiogram from Intact Body, J. A. M. A. **87**:1300 (Oct. 16) 1926.

so weak that the patient was unable to raise the arm at all, and when it had been raised for her, she could hold it at a right angle position only with great effort. The left biceps was weak also, but the patient could flex and extend the left arm at the elbow. The grip of both hands was fairly good, although easily fatigued. Because of the differences on the two sides, we will present the records separately for the right and left biceps and for the two forearms.

Right Biceps: When the right arm was extended, with the hand resting on the patient's lap, records of the type shown in figure 1 *A* were obtained. Isolated small waves occurred at a frequency varying from 5 to 15 per second, at fairly regular intervals, and resembled somewhat those obtained by Wachholder from the biceps of normal persons under the same conditions. In a number of instances, however, we found that the regular series was interrupted by an occasional extra

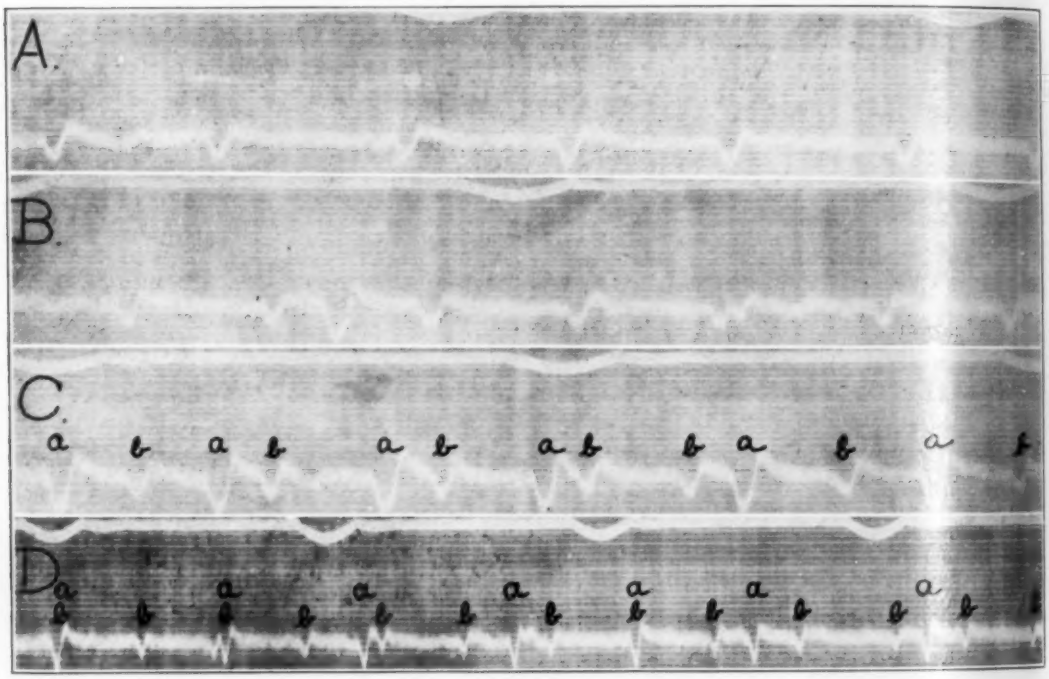


Fig. 1.—Electromyogram from patient with syringomyelia, showing record from right biceps; tension of string 1 m. v. = 1 cm.; time in one-fifth seconds. *A*, record from arm in resting position; biceps under slight tension; frequency of waves, 15 per second. *B*, same as *A*, showing, however, an extra wave of larger amplitude between the regular series. *C*, record showing two series of waves, independent of one another. The frequency of the (*a*) series varies from 12 to 15 per second; that of the (*b*) series from 12 to 20 per second. The two series can be distinguished also by a difference in amplitude. *D*, another record showing two independent series, one at a frequency of from 9 to 10 per second, and one at from 18 to 19 per second. Here again the two series can be distinguished by a difference in amplitude.

wave (fig. 1 *B*), or that the waves were made up not of one, but of two, and occasionally three, distinct rhythms. The two records in figures 1 *C* and *D* showed clearly two independent series of waves, regular in amplitude as well as frequency. In figure 1 *C* one series occurred at a frequency of from 12 to 15 per

second, and the other at from 12 to 20 per second. In figure 1 *D* there is one series at a frequency of from 9 to 10 per second, and one at a frequency of from 18 to 19 per second.

We found also, in agreement with Wachholder and with our own previous work, that the frequency of these waves increased with the tension applied to the muscle. The record in figure 2 *A* was obtained with the arm flexed in a resting position; the second record, figure 2 *B*, just after the patient was told to try to flex the arm; and the third, figure 2 *C*, at the height of the effort. The frequency increased, in this instance, from 4 to 20 per second, and a similar result was obtained in many other experiments. It is important to note that the frequency did not exceed 25 at any time in the records from these muscles, whereas in normal muscles it increases to 75 or 80 per second at the height of a strong voluntary effort. The primary waves, it will be noted, are practically free from Nebenzacken,

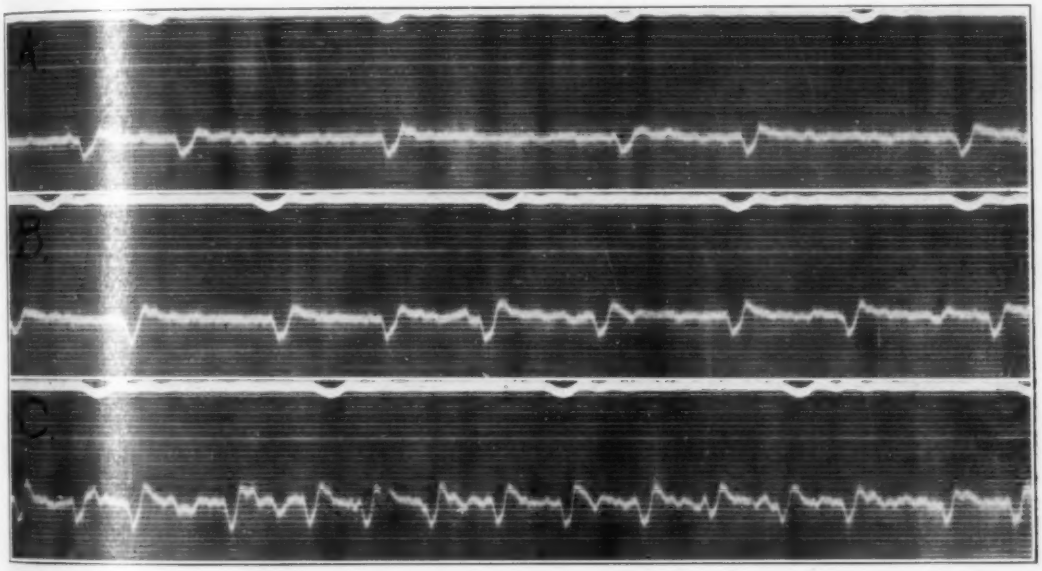


Fig. 2.—Record from patient with syringomyelia showing increase in frequency of the primary waves as the intensity of the contraction increases in right biceps. Right angle flexion at the elbow; isometric contraction against resistance; tension of string 1 m. v. = 1 cm.; time, one-fifth seconds. *A*, arm held in right angle position partly resting against the body; frequency from 5 to 10 per second. *B*, beginning of attempt to lift forearm; frequency from 8 to 11 per second. *C*, at height of effort. Frequency from 15 to 19 per second.

and fairly equally distributed between the individual large waves are the secondary waves, occurring at a rapid rate of 100 or more per second.

Records obtained during passive flexion and extension of the arm are similar to those obtained when a mild voluntary effort was exerted or when the arm was extended in a resting position (compare figures 3 *A* and 2 *A*).

Left Biceps: In the records obtained from these muscles also, the frequency of the primary waves was greatly reduced, but the decrease was not nearly so marked as that in the right biceps. Otherwise, the currents showed the same features, especially the increase in frequency with an increase of tension applied

to the muscle, but the frequency reached 40 in these muscles instead of 20 as in the right arm. The differences in the action currents from the right and left biceps can be seen clearly in the records from passive flexion and extension shown in *A* and *B* of figure 3.

Muscles of the Forearm: The electromyograms from both forearms are characterized by a total absence of large waves. The currents obtained show a high frequency of 150 per second, but their amplitude is small considering the strength of the muscles of the forearm (fig. 3*C*).

CASE 2.—*Progressive spinal muscular atrophy, involving the anterior horn cells of the cervical enlargement and the hypoglossal and ambiguus nuclei.*

Henry M., aged 60, had developed progressive weakness and wasting of the arms, and disturbances in speech and swallowing during the eighteen months

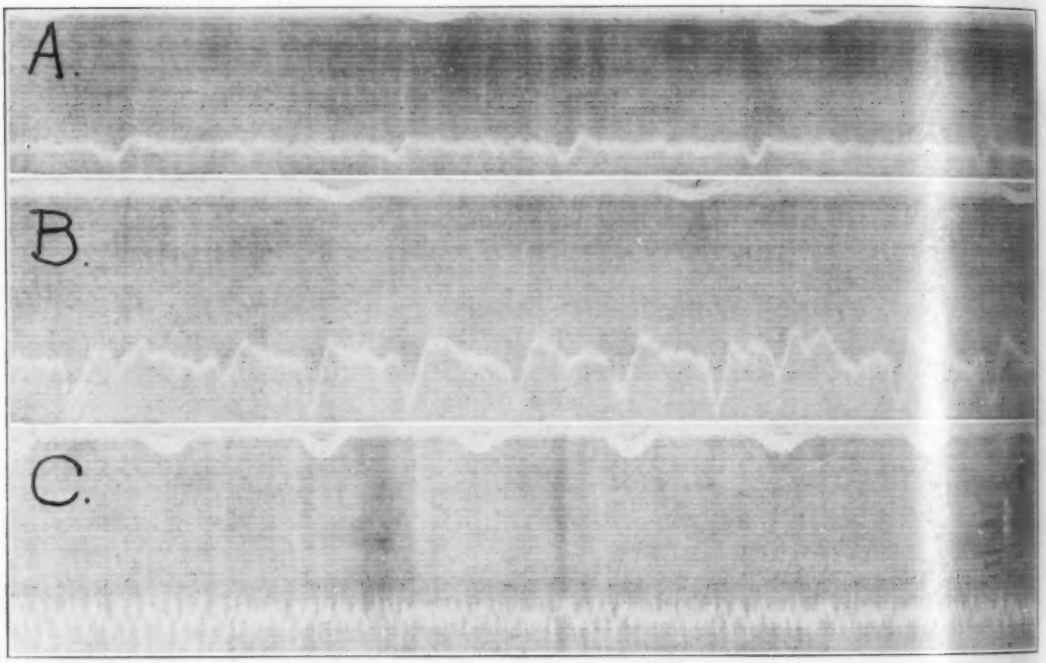


Fig. 3.—Record from patient with syringomyelia; time, one-fifth seconds. *A*, record from right biceps; passive flexion and extension; frequency about 10 per second; small amplitude. *B*, record from left biceps; passive flexion and extension; frequency from 15 to 18 per second. Large amplitude, and irregularities between waves should be noted. *C*, electromyogram from strong voluntary contraction of right forearm

previous to admission. Examination revealed weakness of the soft palate and pharyngeal muscles. The tongue was atrophied and showed fibrillary twitches. There was advanced atrophy of the musculature of the hands, arms and shoulder girdle, and active fibrillary twitchings were seen in these muscles. The extensors of the spine and intercostals were slightly weak, but the legs were strong. Sensibility was normal. In the arms the tendon reflexes were absent, but the knee and ankle jerks were active. The plantar reflexes were flexor in type. The

Wassermann reaction was negative in the blood and spinal fluid. Electrical tests of the affected muscles showed a diminished faradic response but a prompt galvanic response.

Electromyogram.—In this patient records were taken only from the muscles of the right forearm and the biceps; unfortunately none was obtained from the left arm. The muscles of the forearm on the right side were weaker than the biceps and presented an electromyogram which deviated greatly from the normal. In the records in figure 4 *A* and *B*, obtained from a strong, voluntary contraction, the frequency varied between 20 and 30 per second, while the amplitude remained surprisingly constant throughout. The base of the waves was short, in some cases less than 1/110 seconds, so that they resembled the R waves of the electrocardiogram. The frequency of the primary waves increased with the intensity of the contraction just as it did in the patient with syringomyelia, and without a change in amplitude. In the record in figure 5 *A*, the frequency increased

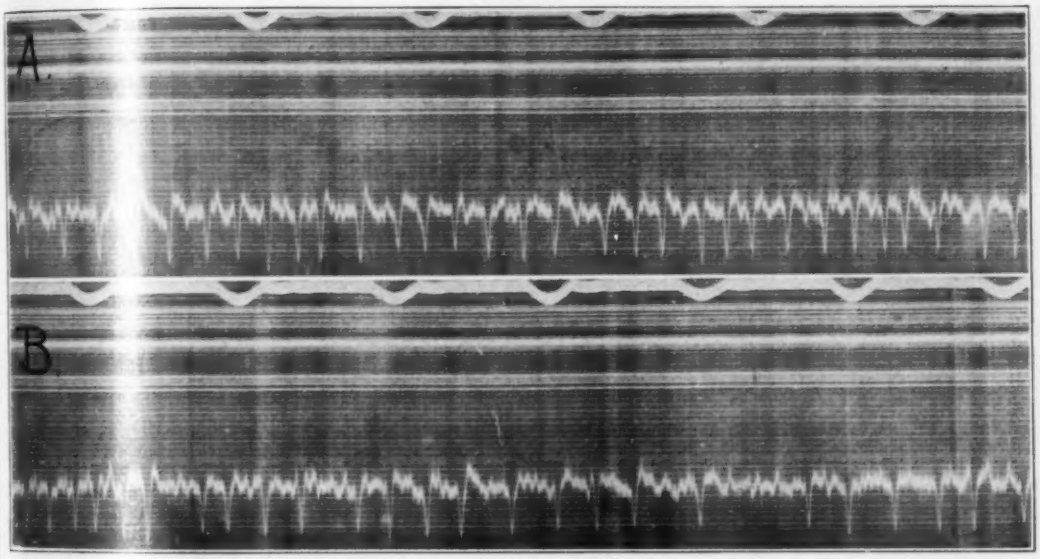


Fig. 4.—Electromyogram from a case of progressive spinal muscular atrophy showing record from right forearm; tension of string 1 m. v. = 1 cm.; time, one-fifth seconds. *A*, frequency from 20 to 30 per second; maximum voluntary contraction; squeezing rod. Numerous secondary waves between primary waves, and short base of primary waves should be noted. *B*, same as *A*, but showing occasional parts of record where primary waves drop out altogether.

within less than a second from 12 to 35 per second. When the forearm of this patient was in a resting position, small isolated currents at a low and irregular frequency were found (fig. 5 *B*) similar to those obtained in the resting muscle in the patient with syringomyelia. We thought that in the patient with atrophy they might have something to do with the fibrillations that were present, but we were unable to confirm this conclusion.

The biceps muscle, stronger and less atrophied than the muscles of the forearm, gave different records. The amplitude of the large waves was smaller here than in the forearm, but the frequency was higher, in some instances 50 per second, and there were many Nebenzacken.

In brief, then, the electromyogram of the patient with progressive muscular atrophy is similar to that obtained from the patient with syringomyelia. The waves occur much less rapidly than in the normal person, and are so clear-cut and well defined that they may be counted without difficulty.

CASE 3.—*Poliomyelitis involving the anterior gray matter in segments C_5 and C_6 on the right and C_8 to T_1 on the left.*

Mary McF., aged 10 years, developed acute poliomyelitis at the age of 5, and both arms were completely paralyzed at first. Examination five years later showed paralysis and atrophy of the right deltoid and spinati, and weakness of the right biceps. The triceps and muscles of the forearm were strong. On the left, the entire musculature of the arm was atrophied, and scarcely any motor power was preserved. The bones of the left arm and the left scapula were much smaller than those of the right. There was no loss of sensibility. The cranial nerves were normal and the legs strong. Tendon reflexes were all lost in the arms except for the right triceps reflex; knee and ankle jerks were active.

Electromyogram.—In this patient records were taken from the biceps and muscles of the forearm in both arms. The left biceps was so greatly atrophied

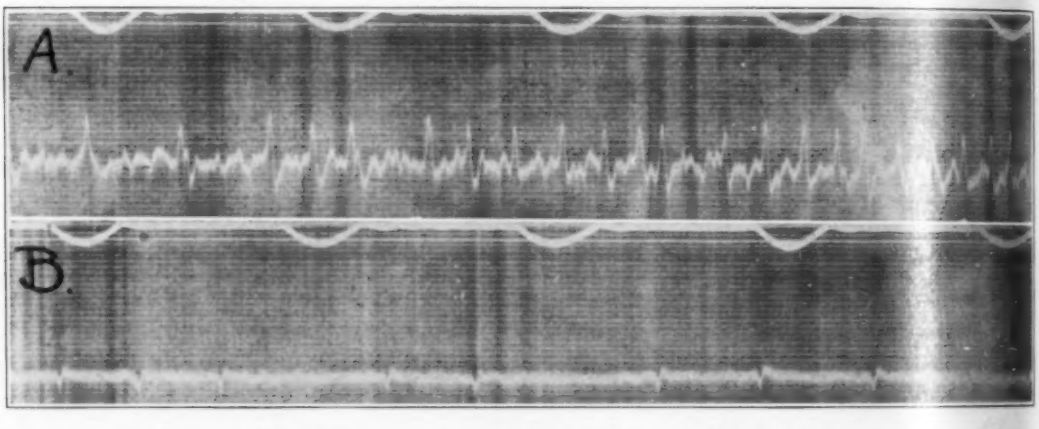


Fig. 5.—Electromyogram from patient with progressive spinal muscular atrophy showing record from right forearm; tension of string 1 m. v. = 1 cm.; time in one-fifth seconds. *A*, record showing increase in frequency of primary waves as the muscle is contracted. At the outset the frequency was 12 per second, and at the height of the effort four-fifth seconds; later it had increased to 35 per second. It is important to note that the amplitude of the waves remained practically constant throughout. *B*, electromyogram from arm in resting position.

that no currents could be obtained from it. The records from the right biceps, which was weak, were similar to those obtained in the two patients already reported. A typical record is shown in figure 6 *A* and *B*. The large waves occurred much less rapidly, while the amplitude of each individual wave was much greater than might be expected. The frequency of the large waves increased as the intensity of the contraction increased, and decreased with fatigue. In the record in figure 6 *A*, obtained immediately after the patient contracted the biceps to a maximum, the frequency rose as high as 45 per second. Ten seconds later (fig. 6 *B*), when the muscle was obviously fatigued but still pulling approximately the same, the frequency was reduced to 20 per second. It is

important to note that, despite the large reduction in frequency, the amplitude remained practically constant. During passive flexion and extension of the arms, the frequency of the waves varied between 25 and 30 per second (fig. 6*C*).

The records obtained from the muscles of the forearm were essentially normal, so far as we were able to determine, with Nebenzacken and secondary waves present throughout.

In three other cases of poliomyelitis in which the patients were examined by us the records were similar to those obtained in this case.

CASE 4 (by the courtesy of Dr. I. W. Nachlas).—*Pseudohypertrophic muscular dystrophy.*

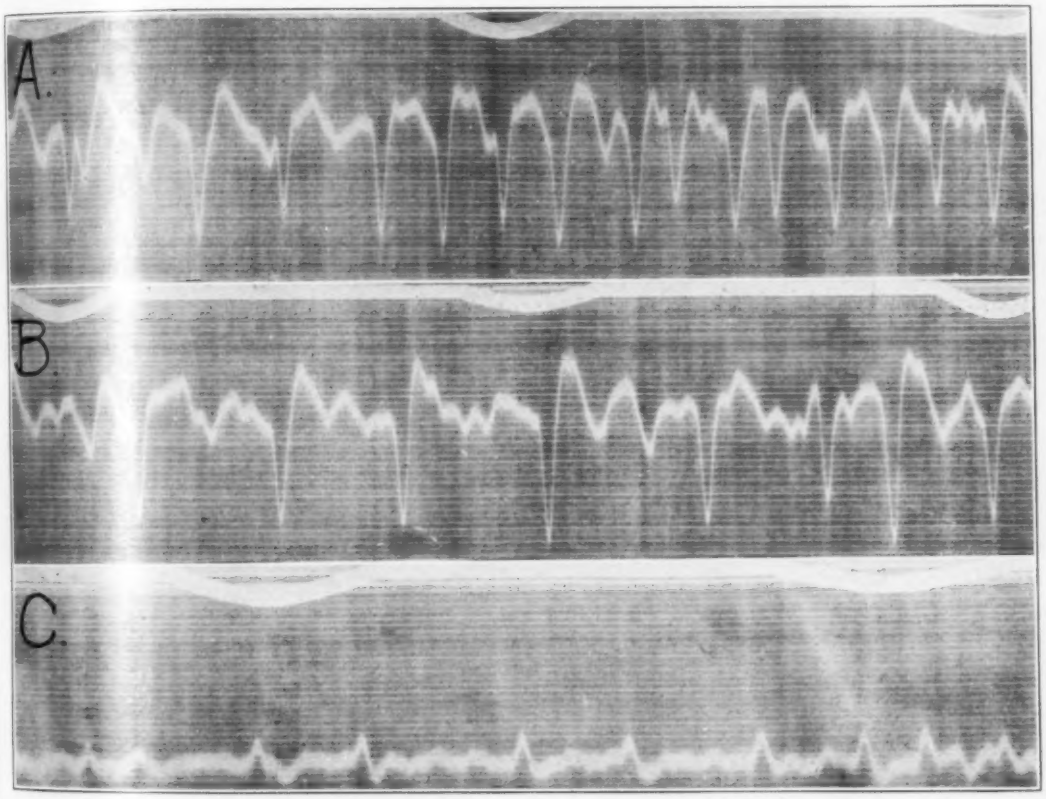


Fig. 6.—Electromyogram from patient with poliomyelitis showing records from right biceps; tension of string 1 m. v. = 1 cm.; time, one-fifth seconds. *A*, record showing action current at height of voluntary contraction; frequency of waves from 35 to 45 per second. Short base of waves and relatively long quiescent intervals between should be noted. *B*, record taken twenty seconds later when the muscles had become fatigued; frequency of waves decreased to about 20 per second. The amplitude remained practically the same. A few of the waves are slightly larger than in the previous record. *C*, record from passive flexion and extension of arm; frequency from 25 to 30 per second.

Oliver H. developed difficulty in walking at the age of 5. This trouble gradually became more pronounced, and at the age of 11 he showed the waddling gait and increased lordosis which are typical of myopathy. The muscles of

the upper arm, shoulder, hip and thigh were all greatly atrophied and feeble. The calf muscles were large and fibrous. All the tendon reflexes were abolished except the ankle jerks. The plantar reflexes were of the normal flexor type. The cranial nerves were not affected, and there were no alterations of sensibility. The patient had one sister who was well at the age of 12.

Electromyogram.—Both biceps muscles were weak in this patient. Typical currents from the right biceps were characterized by a great regularity in the shape and frequency of the primary waves (fig. 7 *A*). The amplitude, on the other hand, varied considerably, but even when at a maximum was not so great as the average amplitude of normal primary waves. The absence of Nebenzacken and secondary waves was striking. Although the frequency fluctuated to some extent throughout the records, from 40 to 70 per second, it was maintained fairly constantly at 55 per second. At the higher frequencies the electromyogram was almost as regular as that obtained from the grasping reflex of the new-born infant. Even when the frequency of the large waves was low (from 40 to 45

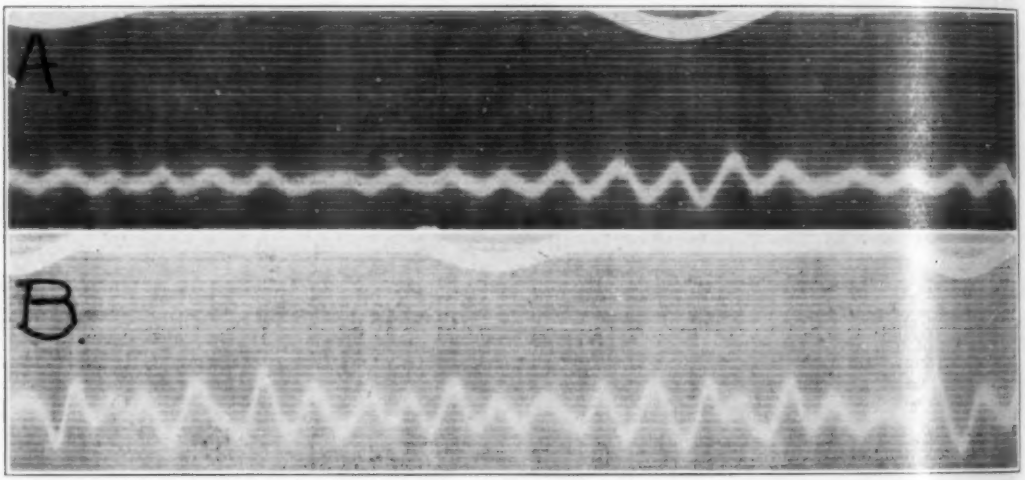


Fig. 7.—Records from two patients with muscular dystrophy; tension of string 1 m. v. = 1 cm.; time in one-fifth seconds. *A*, record from right biceps of Oliver H.; frequency, 55 per second; maximum contraction. *B*, record from right biceps of Alvin M.; frequency, 50 per second. Regularity of waves and absence of Nebenzacken in both records should be noted.

per second), the records of this case contrasted sharply with those obtained in the patients with lesions of the anterior horn cells because of the absence of quiescent periods between the individual waves. The duration of each wave, from one-fiftieth to one-sixtieth second, was unusually long when compared to the average duration of one-hundredth second in the case of progressive muscular atrophy.

CASE 5.—*Pseudohypertrophic muscular dystrophy beginning at 2 years. Typical clinical picture at 8 years.*

Charles F., aged 8 years, an only child of healthy parents, developed difficulty in walking at the age of 2. This weakness slowly increased, until at the age of 8 the child could scarcely stand without support. There was great weakness and wasting of the muscles of the shoulder girdle and upper part of the arm. The

extensors of the spine, and the muscles of the abdomen, pelvic girdle, hip and thigh were also wasted. The calf muscles were large, hard and inelastic. The muscles in the distal segments of the limbs were relatively preserved. Station showed the characteristic lordosis. The gait and method of rising from the floor were typical. The ankle jerks were active; other reflexes were absent. No sensory disturbances were present.

Electromyogram.—Records taken from the biceps muscles, which were weak and atrophied in both arms, were similar to those obtained in the previous case, the only difference being that the amplitude of the waves was even smaller than in case 4.

CASES 6 and 7.—*Progressive muscular dystrophy, Erb's type, in two brothers. Typical clinical picture.*

Alvin and Edward M., aged 15 and 12 years, respectively, developed difficulty in walking at an early age. Two older brothers were similarly affected. Examination revealed all the typical signs of muscular dystrophy with the selective atrophy of the muscles of the shoulder and pelvic girdle, and the well known posture and gait. The calves were not enlarged, but felt rather fibrous. Tendon reflexes were not obtained except for the ankle jerks. No alteration of sensibility could be found. Both patients seemed somewhat mentally retarded.

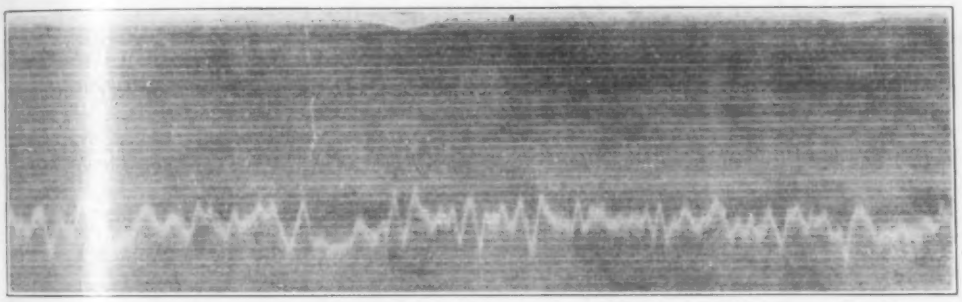


Fig. 8.—Record from tibialis anticus of tabetic patient; strong voluntary contraction; tension of string 1 m. v. = 1 cm.; time in one-fifth seconds. Scarcity of large waves and conspicuous presence of smaller waves should be noted.

Electromyogram.—In both cases the records obtained (fig. 7 *B*) were similar to those described in case 4. The large waves varied in frequency from 50 to 55 per second, and were free from secondary waves and Nebenzacken. There was more variation in amplitude in these records than in the records from the other patients with muscular dystrophy, but the waves were still much smaller than those of normal records.

CASE 8.—*Tabes dorsalis in a man, aged 54. Ataxia and loss of reflexes in all four extremities.*

Charles N., aged 54, developed a penile sore at the age of 22. More than thirty years later, pains and numbness in the legs and disturbances in sphincter control appeared. The pupils were unequal and irregular and did not react to light. There was marked ataxia in the finger-nose and heel-knee tests. The station was unsteady, and the gait was ataxic. There was loss of pain and vibratory and postural sense in the legs. All the tendon reflexes were abolished. The Wassermann reaction was positive in the blood and spinal fluid.

Electromyogram.—The electromyogram from this patient, presented in figure 8, was obtained from a strong contraction of the muscle of the tibialis anticus. The

record was characterized by a general scarcity of large waves, and was relatively flat in view of the intensity of the contraction. Moreover, the smaller secondary waves, with a frequency near 150 per second, were much more conspicuous in this instance than in normal records. The record is clearly different from those obtained from patients with muscular dystrophy and lesions of the motor horn cells.

Similar currents were obtained in several other patients in whom the tabetic process was far advanced, but in cases in which the condition was in an earlier stage the records could not be distinguished from those of normal persons.

SUMMARY OF EXPERIMENTAL RESULTS

On the basis of the records described we may say that in patients with lesions of the motor horn cells, syringomyelia, progressive muscular atrophy and poliomyelitis, voluntary contractions are accompanied by an electromyogram different from the normal, showing the following characteristics: (1) a reduction in frequency of the primary waves from 50 to 75 to from 5 to 10 per second; (2) a relatively large amplitude in view of the feeble contraction of the atrophied muscles; (3) primary waves with a small base (in many instances less than one one hundred and twentieth second, as compared to the base of one-sixtieth in most normal records), with long flat intervals between the individual waves, and (4) a remarkable freedom from Nebenzacken. Such records, according to our knowledge, are never obtained in normal persons.

Electromyograms from patients with muscular dystrophy, whose muscles are also atrophied, but because of a local non-nervous condition, show the following characteristics: (1) regular primary waves, with a frequency varying from 40 to 60 per second; (2) primary waves with a broad base with no quiescent intervals between, and (3) few Nebenzacken. Similar curves have been obtained occasionally in normal persons, but rarely.

The electromyogram from tabetic patients shows a wide range of variation. In mild cases it cannot be differentiated from those of normal persons, except that the primary waves occur relatively less frequently than usual, and that the secondary waves are more prominent. In advanced cases, the primary waves disappear altogether and the remaining currents show a high irregular frequency.

Action currents from these three different types of cases can be distinguished from one another and from the normal readily, simply by inspection, without the measurement or counting of waves.

COMMENT

What light do these results throw on the origin and significance of the different parts of the electromyogram from voluntary contractions? In view of our previous investigations we were especially interested in determining to what extent they would lend themselves to our theory

of the proprioceptive origin of the primary waves. Before the evidence for and against this view is presented, it is necessary to outline again the chief characteristics of the proprioceptive impulses, as determined by Adrian and Zottermann [8] in their experiments in which the action currents from the afferent nerves were recorded when the muscle was stretched. In the intact muscle with a number of proprioceptive end-organs functioning the results were not clear-cut, since numerous irregular currents were present at a frequency of from 100 to 400 per second. It was found, however, that by stripping the muscle carefully, little by little, a stage could be reached when apparently only one or two end-organs remained. The action currents from such a nerve-muscle preparation were simple, consisting usually of only one series of regular waves, increasing in frequency from 5 to 100 per second as the tension applied to the muscle increased, and decreasing as the tension was prolonged. Occasionally, records were obtained in which two independent series with different frequencies were present, apparently when two end-organs were still functioning. Besides these characteristics, the action currents showed a constant amplitude, in keeping with the "all or none" principle, regardless of the tension applied to the muscle.

With these facts in mind we may consider the characteristics of the primary waves found in the present experiments. In the three patients with lesions of the anterior horn cells, usually only one series of large waves was present, and it will be seen that they showed practically the same characteristics as have been described for the proprioceptive impulses. 1. Their frequency increased with the tension applied to the muscle from 5 to 50 per second and decreased with the duration of the contraction. Most workers have assumed hitherto that the frequency of the primary waves of the electromyogram remained constant regardless of the condition of the muscle. 2. The amplitude of the waves remained practically the same, and neither increased with the tension applied to the muscle nor decreased when the tension was released. 3. Occasionally, in our records two independent series of primary waves with different frequencies were present, just as they were in the records of Adrian and Zottermann. Such a striking similarity between the primary waves and the proprioceptive impulses, therefore, seems to justify the interpretation that the primary waves are produced by the proprioceptive impulses. This conclusion cannot be accepted, however, without further consideration, because of the fact that in all of the six cases with a lesion of the motor horn cells reported in this paper, as well as in the two cases described by Weizsäcker,[7] the reflexes were apparently absent. In other words, then, the reflex arc was interrupted, so that it was impossible for proprioceptive impulses to manifest themselves in the records. We believe that the reflex pathway was not absolutely transected, and we shall recur to this contingency later. First, however, we must consider

what is deduced in the present experiments from an assumption of a reflex origin for the large waves.

If the primary waves arise in the reflex arc but not in the proprioceptive impulses, then the only place of origin remaining is in the motor horn cells. Weizsäcker, and Dusser de Barenne and Brevée [9] have attributed an inherent or autochthonous rhythm to these cells which, they believe, is responsible for at least part of the large waves of the electromyogram from voluntary contractions. Weizsäcker believes that the large waves with the slow 15 to 18 per second rhythm found in his pathologic cases do not really correspond to the primary waves of the electromyogram, but appear only when the impulses from the proprioceptive end-organs have been eliminated. Under normal conditions, when the reflex arc is intact, the proprioceptive impulses passing through the motor horn cells break this slow inherent rhythm into the faster, more irregular rhythm found in voluntary contractions.

Against Weizsäcker's conception it may be said in the first place that the rhythm present in the pathologic cases is not fixed, but may vary from 2 to 50 per second with the size of the contractions; and the transition from the lower to the higher frequencies is perfectly smooth. Weizsäcker apparently did not study the effect of changes in muscular tension on the frequency of the large waves. In the second place, if this reduction in frequency of the large waves is due to the interruption of the reflex arc, then we should expect to find a similar record in tabetic patients, in whom the posterior roots are degenerated. The records from tabetic patients, however, are different from those obtained from the patients with lesions of the motor horn cells. It is not the interruption of the reflex arc, therefore, that is important.

We believe that in the various cases with extensive lesions of the motor horn cells already discussed, the reflex arc probably was not entirely interrupted. A few fibers were still functioning, perhaps, but not enough to give rise to a twitch sufficiently large to be detected clinically This theory is upheld by the fact that in all our patients with the lesions of the motor horn cells, we obtained the slow rhythm of the primary waves in response to passive movements of the arms; that is, through reflex stimulation of the proprioceptive end-organs, produced by the stretching of the muscles. The conditions, then, as we have already pointed out, would be similar to those present in the experiments of Adrian and Zottermann. They decreased the number of functioning fibers by stripping the muscle, whereas in the present experiments the same end was attained through lesions of the motor horn cells, so widespread as to leave only a few fibers intact.

The low frequency of the primary waves, in comparison to their frequency under normal conditions, was due, we believe, to the fact that it was not possible for enough impulses to pass through the motor horn

cells to produce an average strong contraction, and thus a higher frequency of the proprioceptive impulses. We would also explain the simplicity of the waves, their narrow base and their freedom from Nebenzacken on the basis of our assumption that only a few pathways through the motor horn cells were functioning.

The views of Dusser de Barenne and Brevée differ from those of Weizsäcker only as regards the frequency of the inherent rhythm of the motor horn cells. They believe that it lies between 50 and 70 per second, and that this rhythm, like Weizsäcker's, becomes visible in the records after the proprioceptive impulses have been removed. Under ordinary conditions, when the arc is intact, the proprioceptive impulses, passing through at a much higher rate, entirely obscure the inherent rhythm. Dusser de Barenne and Brevée arrived at this conclusion through the following experiments: When the exposed surface of the spinal cord of cats and frogs was touched lightly with a cotton swab, the action currents from the resulting contractions of the legs were similar to those obtained from voluntary contractions. When the cord was painted with procaine hydrochloride before it was stimulated, however, the action currents gradually became more regular and diminished in number, at the same time, of course, that the strength of the contraction decreased. In this way, a stage was reached finally at which only a regular rhythm remained, with a frequency between 50 and 70 per second. The authors believe that at this stage the sensory mechanisms were entirely excluded, and that the resulting rhythm was produced by the motor mechanism in the cord.

We may note that there is a marked similarity between the records of Dusser de Barenne and Brevée and those obtained in the present paper from patients with muscular dystrophy, but we do not see just how these facts can be brought into agreement. It may be that we are dealing with two separate rhythmic processes, one a proprioceptive mechanism and the other an inherent rhythm of the anterior horn cells. The presence of a rhythm of the anterior horn cells of from 50 to 60 per second may help to bring the proprioceptive impulses into phase, since at this rate the action currents are always regular and free from Nebenzacken.

The reduced rate of the primary waves in the three patients with lesions of the anterior horn cells was probably due to the fact that the transmission of impulses from the higher centers through the anterior horn cells was greatly hindered by the disease processes and that for this reason the muscle could not be brought into full tension. The reduced rate would then result from the slight tension. In the patients with muscular dystrophy, on the other hand, what remained of the muscles could be brought into full tension, so that we found in them the usual high frequency action currents.

SUMMARY

1. Electromyograms from voluntary contractions in syringomyelia, progressive muscular atrophy and poliomyelitis are all similar, and all deviate widely from the normal. We have suggested that these peculiar currents are characteristic for lesions of the anterior horn cells, common to these three different diseases. They all show an atrophied condition of the muscles, but this is not the important factor, since a similar electromyogram is not found in other cases, such as muscular dystrophy, in which the atrophied condition is not produced by lesions of the anterior horn cells.

2. The frequency of the primary waves is markedly reduced (to from 3 to 5 per second in some instances), but, as in normal records, it increases with the intensity, and decreases with the duration of the contraction.

3. The amplitude of the waves is large considering the atrophied and enfeebled state of the muscles, and remains practically constant, regardless of the intensity of the contraction.

4. Occasionally two separate series of waves at low frequencies can be seen in the records.

5. The primary waves are of much shorter duration than in normal records, often less than one one hundred and twentieth second, so that they look like the R wave of the electrocardiogram.

6. The records from patients with muscular dystrophy also deviate from the normal, but in a different way. The primary waves are much more regular both in frequency and in amplitude than is usual in normal persons, and they are almost entirely free from all irregularities, either Nebenzacken or secondary waves.

7. The origin and significance of the different parts of the electromyogram were discussed on the basis of the records obtained from the pathologic nerves and muscles. It was shown that the records bear out fairly closely the theory that the primary waves are produced by the proprioceptive impulses, as presented by Richter in a previous paper. It was shown, however, that our present observations are also, at least partially, in agreement with the view of Weizsäcker and Dusser de Barenne, who arrived, from different angles, at the conclusion that the primary waves are somehow produced by an inherent or autochthonous rhythmic activity of the motor horn cells.

HAS THE PEDIATRICIAN ANY PLACE IN THE MENTAL HYGIENE MOVEMENT?*

BRONSON CROTHERS, M.D.
BOSTON

The campaign for mental hygiene has been vigorously conducted for the last decade. By this time, it should be possible for every interested physician to formulate an opinion about the movement and to adjust himself to it. This adjustment should be particularly easy if the repeated statement that mental disease is like tuberculosis is valid.

The pediatrician, of all physicians, should decide on his attitude, and it is from the point of view of the pediatrician that I have written this article. Pediatricians like to think of themselves as general practitioners who supervise humanity during the period of growth. On the whole, they have looked with a good deal of approval on the vigorous generation which is now taking control of the world. In a way, the young adults of the present are their first-born, since pediatrics as a recognized specialty is only about 30 years old. The young people of today are demonstrably less rachitic, have fewer infections and perhaps are better equipped to meet the physical perils of life than their predecessors. In modest moments, pediatricians admit that pasteurization of milk, the discovery of vitamins and the utilization of sunlight are not pediatric discoveries, but they have thought of themselves as inspired borrowers and adapters of promising discoveries. Their general idea is to look over and modify any new discovery so that their patients may get the full benefit of it. Naturally they try to make their contributions, of some of which they are legitimately proud.

The psychiatrists have suddenly shattered their complacency by announcing that the body is simply a part of the person, and that psychiatrists are the only true general practitioners, all others being practitioners of a part of medicine. Moreover, a certain group of them announce that the period of childhood interests them greatly, and that they purpose to take over the supervision of children. The average pediatrician did not know what to do about this unexpected challenge a few years ago, and some have spent a good deal of time trying to get at facts that would enable a proper decision to be made.

PROBLEMS OF THE PEDIATRICIAN

The easiest solution, of course, is to deny the validity of the psychiatric conception and go on with Schick tests and studies on the influence of cod liver oil on bones and of ultraviolet light on milk, giving

---

* Read at the Fifty-Third Annual Meeting of the American Neurological Association, Atlantic City, N. J., May 25, 1927.

optimistic opinions on whatever mental problems come. Unfortunately, this evasion is widely practiced. It is, clearly, extremely easy to quote passages from some of the more fantastic bits of psychiatric literature and to poke fun at the whole subject. As a permanent refuge, ridicule of this sort is decidedly weak, for more and more people realize that psychiatry is a reputable specialty.

Another method is to accept at their face value the psychiatric primers which are constantly sent to pediatricians. Since these elementary volumes are planned to excite interest and to promote understanding among the laity as well as among physicians, it is obvious that the text is simple, and that many of the directions can be easily followed. The only trouble is that following such elementary directions often leads into, rather than out of, trouble. Any one who deals with children and their parents either as a pediatrician or as a psychiatrist meets people who have suddenly become convinced that their children are suffering from urgent inferiority complexes, that teachers are incapable of establishing helpful libidinous transfers, or that the evolution of polymorphic pervertism is not going well. As a medium for scientific exchange of ideas among experts, such terms are valid; as diagnoses among untrained people, they seem to be of dubious value. I believe that the pediatrician is justified in rejecting the psychiatric vocabulary until he establishes a background.

Another suggestion is that pediatricians should become effective psychiatrists. This appeals at once. Within a brief specialized life, pediatricians become fairly good chemists, rather satisfactory epidemiologists and amazing dietitians. If mental disease is like tuberculosis, one feels justified in taking up psychiatry without misgivings. It is at this point that I find psychiatrists most confusing. Any one who reads or listens to the propaganda for mental hygiene and checks it against the scientific psychiatric literature finds serious discrepancies and contradictions.

For instance, I heard a speaker state before a lay audience that 50 per cent of the cases of insanity for which the patients have been committed to institutions could have been prevented if the patients had been managed properly; he said that the condition could have been prevented by "technic." I have waited in vain to hear any one claim before a medical audience that "technic" prevents any form of insanity. Then I found this statement:[1]

> When a mentally defective woman has one child before she is segregated, while there is no increase in the number of mentally defective individuals in the next generation, still there is no decrease; and when she has more than one child, the total number of mentally defective persons in the next generation is increased.

1. Wolfe, M. M.: Mental Hygiene, **7**:342, 1923.

Yet in any discussion before any critical medical audience such a statement would be challenged by every one present.

The attempt to tell people more than they can possibly be expected to understand is illustrated in the September, 1924, issue of the *Monthly Bulletin of the Massachusetts Society for Mental Hygiene*. In order to clarify the attitude of the public, the following series of definitions is presented:

In these days of public interest about abnormal minds and queer personalities it is only natural that the layman should feel confused as to what many of the new terms are all about.

Psychiatry, for example, is a tongue twister. Pronounced sy-ky-at-ree, it refers to a branch of medical science dealing with disorders of the mind.

Its exponent is a psychiatrist, a graduate physician of a recognized medical school, who has superimposed on his regular medical course additional years of training in this specialty. Formerly he was called a *brain specialist* or perhaps a *neurologist*.

A *neurologist*, however, is a physician who deals with the physical diseases of and injuries to anatomical nerve structures.' Thus for locomotor ataxia with its damaged central nervous system, or perhaps for a brain tumor, the neurologist would be called, while for hysteria or for dementia praecox (a form of so-called insanity) where no physical damage to nerve structures can be discovered and where the symptoms are chiefly mental, the psychiatrist would hold sway.

Then there is the *alienist*. He, too, is a physician who may be either neurologist or psychiatrist. But the alienist's chief interest lies in the medicolegal aspects of the case.

Psychosis is merely the medical term for any of the definite insanities.

A *psychologist* is seldom a physician. He studies man's feelings, his intelligence and behavior, whether he be sick or well. The psychiatrist studies man's mental sickness and the mental, physical, and environmental causes of it. The psychologist is interested in how man adjusts to new situations, the psychiatrist in why he does not adjust.

Schizophrenia, pronounced skeez-o-freen-ee-ah (its adjective is schizoid, "skeez-oyd"), is most perplexing of all. It refers to a splitting off or a detachment of one part of the personality from another. Day-dreaming is a sort of example where one part of the individual's mind is so wholly absorbed in building his castles in Spain that only dimly is he aware of what goes on around him in the world of reality. In cases of dementia praecox, a serious mental disease affecting young people, hours, sometimes days or even weeks, are thus spent completely wrapped up in a day-dream world of the patient's own making, entirely oblivious of reality.

Entirely aside from the extreme brevity and inaccuracy of the definitions, it seems to the pediatrician that it is hardly conceivable that an unfortunate layman, ignorant of the name of the science of psychiatry, could be brought in one small column to profitable discussion of the technical mechanism of schizophrenia.

Besides such reckless statements, there are articles telling the pediatrician what to read in order to understand mental mechanisms. I may

be dull, but a list of articles suggested in *Mental Hygiene*,[2] seems a bit appalling and unlikely to give a clear idea of anything in particular, yet it is recommended that college students read this material during six weeks study of mental hygiene. The instructor, not a medical man, took up Dr. Morton Prince's "The Unconscious" and "The Dissociation of Personality" and left the students to struggle alone with: (1) Brill's "Fundamental Conceptions of Psychoanalysis," (2) Coriat's "Abnormal Psychology," (3) Jackson and Salisbury's "Outwitting Our Nerves" and articles of various types, ranging from Myerson's on "The Nervousness of the Jew" to Paton's "Psychology of the Radical" and Gilbert's "Amnesia—the Subconscious Warehouse."

I have more respect for Dr. Prince and Dr. Brill and the rest of the authors than I have for the man who thought that because his students said they liked the books the effort was well advised. The inference that the time spent in experiments was to be cut down because it was voted dull by most, and that spent on abnormal psychology was to be increased because it was voted exciting, seems a bit ill advised.

It would be possible to pick out many irritating articles and other minor matters from the generally admirable publications of the National Committee for Mental Hygiene and other societies if they proved anything. Obviously they do not. The only question of importance is whether a pediatrician can get the essentials of authentic mental hygiene out of casual reading of literature which is devised to interest laymen as well as physicians. I doubt the possibility. It is equally out of the question for most pediatricians to travel the long road to real understanding of the conflicting psychoanalytic schools, and any timid sight-seeing trips seem to some of those who have tried them not to be of great value.

It remains to test the theory that psychiatrists have assets other than complicated and disputed technical methods and elaborate vocabularies. If these less advertized goods can be acquired, the pediatrician will have made precious gain. It is clear, I think, that the useful psychiatrist is not a mere technician; neither is he made in a day or a year. His fundamental assets are, I should say, the following: (1) wisdom, persistent curiosity and tolerance; by experience he acquires an insight into the mental difficulties of mankind; (2) the acquisition of various technical methods of study and vocabulary which enable him to formulate ideas and to save time in discussion with others of similar interests and comparable training. In individual cases, he knows how rigidly he must explain and reexplain his meaning before the patient or his friends understand.

To become a psychiatrist of this type is obviously out of the question for the pediatrician in active practice. A dangerous alternative is to

2. Laird, D. A.: Mental Hygene, **7**:27, 1923.

borrow the vocabulary and some of the technic and to start without the fundamental virtues and assets. I have come to the conclusion that the real difficulty is that many of the leaders of the mental hygiene campaign, both inside and outside of the National Committee, are dangerously self-depreciatory. Instead of saying arrogantly that they are wise, tolerant and experienced investigators of human conduct, they pass lightly over their personal virtues, which are their real claim to eminence, and advertise their technic and their vocabulary.

The insistent suggestion that mental disease is like tuberculosis seems to me another product of the supermodesty of the psychiatrist. After all is said about the variety of symptoms and of pathologic processes in tuberculosis, the study of the disease is simplicity itself compared to the problems that confront those who attempt to study and control human nature.

Much has been written with the hope that the stigma of mental disease could be removed. Vast and encouraging progress has been made. But it seems to me and to many of my pediatric associates that the effort is sometimes too insistently pursued. The habits of the child are matters which properly concern the psychiatrist, but many mothers are found who acquire, naturally enough, the idea that tantrums are mental disease in a nascent state and that the logical end of maternal tenderness is suicide. A leading editorial, published in a rural journal in New England and said to be signed by Dr. Alfred Adler of Vienna, states:

> Mother love is the one thing, more than any other except physical handicap, that mars the character of many children, yet any mother can avoid that catastrophe if she learns the behavior tenets.
>
> Mothers are too likely to destroy, or minimize courage in their babies. The child learning to walk totters, reaches out for mamma and falls in her arms instead of on the floor. Depending on mamma. The child gets hurt, a bump. Mamma soothes. Mamma does everything. The result is an inferiority complex. The child feels the mother's superiority, becomes a coward, sometimes. Cowardice is a vicious trait. It stops proper development of the social character, leads to suicide. Suicide is cowardice.
>
> Then there is the inferiority complex that is developed in the first born because of the second born. For maybe one year, two years, three years, the first born has been the center of the home, receiving all attention. Unprepared for the coming of the second child he finds his place usurped. Hurt, not understanding, the first born becomes spiteful, mean, sullen.
>
> The later children are the best equipped. They never get to be the center of the home; mother love isn't lavished upon them. They have to fight, they have to develop independence. And how often, indeed, we see the last born outstrip the older brothers and sisters.

I realize fully that the National Committee for Mental Hygiene has no responsibility for this masterpiece, but consider for a moment the effect of such writing on the people to whom it is addressed. As far as

I know, and I am a fairly consistent reader of that particular local weekly and spend much of my time in the neighborhood, there is no reason to suppose that any of the mothers have the slightest understanding of the conflicting ideas about the inferiority complex. Does such a publication simplify or accentuate the efforts of those who are trying to clear up misconceptions about insanity?

In a more sophisticated setting, do the lectures of Dr. Rank to social service workers clear up doubts or increase them? The trouble seems to me to arise in a theory that instruction makes experts. The psychiatric social worker or the graduate of a series of psychiatric lectures is assumed to be able to digest and to evaluate technical conceptions which are subjects of doubt to the elect.

So far I have simply expressed the bewilderment of the pediatrician. I should like to suggest, more for discussion than anything else, my individual method of escape from the fascinating traps which seem to close about the pediatrician who considers mental hygiene.

METHOD USED BY AUTHOR

I spend much of my time dealing with children who have physical disabilities due to organic lesions of the nervous system. Since the ordinary mortal regards the mind and the nervous system as somewhat related, I see a good many children with mental problems. About two years ago, a social worker trained in psychiatry was added to our staff at the Children's Hospital, where an effort is made to do the best that we can with the various mental difficulties that are presented.

Many of the problems are referred to one of the established psychiatric clinics. For a while most of them were referred, but as our interest increased, the transfers were followed up; roughly 50 per cent of the cases were lost on the way. Remembering that it had been stated that 50 per cent of mental disease could be prevented and that childhood was the time to do it, the initial waste seemed appalling.

Next, the establishment of a psychiatrist in our clinic was tried, rather half-heartedly on both sides. It was at once clear that any permanent arrangement of this sort would require more workers, and the relatively elaborate plans seemed to involve more financial aid than could be asked for from the trustees of the institution.

The staff therefore settled down to work out a plan suited to our limited mental and financial resources. The social worker cooperated with me valiantly. She agreed to use only words that we both understood and which either of us could explain to a neutral pediatrician. In every complicated case one or both of us talked with the child, with both parents and with the teacher. To me this was a most unusual procedure. I had hardly realized how few fathers and teachers a

pediatrician comes in contact with, except under the totally abnormal conditions of urgent physical disease.

When we began to have more or less confidence in ourselves, we found, just as the psychiatrists had predicted, that a great many people were finding it hard to bring up children, and that if all the people who were bringing up an individual child could be assembled, either as a group or in series, many difficulties could be straightened out. As time has gone on, more and more technical terms have crept into our conversation, but we are becoming convinced that our progress, and what we hope is our success, is due to sustained interest and increasing experience rather than to the thin veneer of technic which is available to us.

In a way, of course, we are doing psychiatric work. We have unquestionably borrowed many of our methods, but we are unwilling to accept the name of psychiatrists. This is due to our sense of pride. We would prefer to be regarded as rather satisfactory pediatricians than as demonstrably poor psychiatrists. It is hoped that a useful conception of the whole child may be arrived at by adding the study of mental processes of a limited sort to a fairly adequate conception of somatic disease.

From the pediatric point of view, an interest in mental processes is obviously all that keeps the specialty within the field of general practice; therefore, pediatricians should eagerly cooperate with psychiatrists.

Psychiatrists might well make serious efforts to fit pediatricians into a scheme of mental hygiene. A barrier of superficial misunderstanding has arisen. The issue seems to be clear: If mental hygiene is a separate branch of science for which technical tools are of predominant value, few pediatricians will enter it; if it is one of the most vital parts of the practice of medicine, it will appeal to the best pediatricians as a new, vividly interesting task to be entered on with fear but also with enthusiasm.

Psychiatry as a dignified specialty does not need any defense from scoffers, but it does need protection from those who suggest that it can be divorced from medicine and placed in the hands of technicians.

THE ETIOLOGY OF MIGRAINE*

J. CLARK MOLONEY, M.D.
DETROIT

It has been suspected that the substratum underlying the "volcanism" of migraine is identical with the process responsible for epilepsy and such allergic phenomena as asthma, hay-fever and urticaria,[1] and that the respective clinical manifestations are but regional displays of one and the same mechanism.[2] Further, it has been intimated, that this mechanism is hereditary,[3] being transmitted according to the principles of Weismann after the theory of Mendel.[4] But it is known to be more than hereditary. It is an intrinsic condition, and, like gun-powder, requires a detonator to bring about an explosion. These detonating substances are countless, and Gordon, in speaking of the spark that fires the load, facetiously observed, "Who frowed dat brick?" [5] Pollens, dander, bacterial proteins, biliary infection,[6] catabolites and colonic residue have been impugned in turn.

First blood is drawn from the statisticians, who have attempted to mine and to assay the underlying vein and to determine the prevalence of these clinical entities. A measure of their difficulties is indicated in table 1.

Auerbach believed migraine to be the most prevalent of all types of headache.[7]

Statistics are not available on the percentage of migraine or epilepsy in a given locality, state or country.[8]

The difficulty lies in the diagnosis, and although asthma, hay-fever and even epilepsy have certain diagnostic earmarks by which they are

---

* From the Division of Neuropsychiatry, Henry Ford Hospital.

1. Kämmerer, Hugo: Ueber Pathogenese und Therapie der Migräne, München. med. Wchnschr. **72**:633 (April 17) 1925. Miller, J. L., and Raulston, B. O.: Treatment of Migraine with Peptone, Evidence of the Anaphylactic Character of the Seizure, J. A. M. A. **80**:1894 (June 30) 1923.

2. Liveing, E.: On Megrim, Sick-Headache, and Some Allied Disorders: A Contribution to the Pathology of Nerve-Storms, London, J. & A. Churchill, 1873.

3. Möbius, P. J.: Die Migräne, Wien, A. Hölder, 1903.

4. Buchanan, J. A.: The Familial Distribution of the Migraine-Epilepsy Syndrome, New York M. J. **113**:45, 1921; The Study of the Hereditary Factors of Epilepsy, Minnesota Med. **3**:536 (Nov. 20) 1920. The Mendelianism of Migraine, M. Rec. **98**:807 (Nov. 13) 1920.

5. Gordon, A. H.: Some Aspects of Migraine, Internat. Clinics **1**:120 (March) 1924.

6. McClure, C. W., and Huntsinger, M. E.: Observations on Migraine, Boston M. & S. J. **196**:270 (Feb. 17) 1927.

7. Auerbach, Siegmund: Headache, Oxford University Press, 1913.

8. (Letter on) Migraine and Epilepsy, J. A. M. A. **81**:1809 (Nov. 24) 1923.

known, migraine offers a more complex problem. Migraine, as is known, is a symptom-complex characterized by an agonizing headache of the boring or bursting type, first localized over one or the other parietal region but rapidly involving one side of the head or the whole head, it is associated with nausea and vomiting and often with hemianopia and even with mania. It is frequently preceded by an aura of scintillating scotomas and fortification figures, ascending paresthesias of the opposite arm or leg or formication of the opposite side of the tongue. The condition is transmitted as a recessive character,[9] usually insinuating itself within five to ten years of puberty; the attacks are irregular in frequency and severity, lasting for from a few moments to several days, and are followed by a deep sleep of from three to four

TABLE 1.—*The Prevalence of Migraine, Psychogenic Headache, Epilepsy and Allergy*

Observer	Number of Cases	Headache	Migraine	Psychogenic Headache	Epilepsy	Allergy
Peterman: Am. J. Dis. Child. **32**: 416 (Sept.) 1926	Pop. U.S.A. (estimated)	....	....	....	0.31%	....
Duke: Allergy, Asthma, Hay Fever, Urticaria and Allied Manifestations of Reaction, St. Louis, C. V. Mosby Co., 1925	Pop. U.S.A. (estimated)	....	....	....	....	15%
Jelliffe: Modern Medicine, Philadelphia, Lea & Febiger, 1915, vol. 5, p. 709	3,000	98%	50%	....	....	....
Moloney	428	38.3%	8.4%	8.9%	....	....
Moloney	347	....	....	....	....	17.8%

hours' duration, after which the person is incapacitated for from several hours to three or four days, depending on the severity of the attack. Psychogenic headache is most often confused with migraine because of the personality of these sufferers, their tendency to fabrication, their notorious pylorospasm inducing nausea and vomiting and the hereditary tendency of neuroticism.

Innumerable statistics have been compiled purporting to prove or to disprove the migraine or epileptic diathesis, and more particularly their so-called common origin. A few of these are given in table 2.

Further, Gowers[10] called attention to the frequent association of migraine and asthma in certain families. Abel[11] unwittingly used a

9. Guyer, M. F.: Being Well-Born, Indianapolis, Bobbs-Merrill Company, 1920.

10. Gowers, W. R.: A Manual of Diseases of the Nervous System, London, J. & A. Churchill, 1886, vol. 2.

11. Abel, G.: Die Verwendbarkeit des Migräneserums "Bohnstedt" in der Gynäkologie, Deutsche med. Wchnschr. **47**: 1229 (Oct. 13) 1921.

foreign protein when he used placental extracts in the treatment of patients with migraine. Pagniez, Vallery-Radot, and Nast [12] and Rohrer [13] believed migraine to be an anaphylactic reaction. Kämmerer [14] used the term "allergic pathogenesis" in speaking of the disease. Liveing [2] referred to certain cases of asthma as being transformations of migraine. Möbius [3] felt that asthma frequently develops in patients with migraine. Block [15] stated that migraine and angioneurotic edema

TABLE 2.—*Frequency of Epilepsy, Migraine or Allergy in Family History*

Author	Number of Cases		Family History		
			Epilepsy	Migraine	Allergy
McClure: Boston M. & S. J. **196**: 270, 1927	Migraine.....	8	....	62.5%	....
Peterman: Am. J. Dis. Child. **32**: 416, (Sept.) 1926	Epilepsy.....	..	9%	21%	....
Spratling, quoted by Peterman	Epilepsy.....	..	16%	....	....
Turner, quoted by Peterman	Epilepsy.....	..	37%	....	....
Dejerine, quoted by Peterman	Epilepsy.....	..	21.2%	....	....
Binswanger, quoted by Peterman	Epilepsy.....	..	11%	....	....
Miller and Raulston (footnote 1, reference 2)	Migraine.....	25	4%	68%	8%
Smith: J. Michigan M. Soc. **24**: 478, 1925	Epilepsy.....	607	2.4% ascendants†	....	....
Burr, quoted by Smith	Epilepsy.....1,449		2.3% ascendants	....	....
Thoms, quoted by Smith	Epilepsy.....	138	5.8% ascendants	....	....
Thoms, quoted by Smith	Epilepsy.....	138	1.8% descendants†	....	....
Stuchliks, quoted by Smith	Epilepsy.....	176	4.1% ascendants†	....	....
Hubbel: J. A. M. A. **51**: 480 (Aug. 8) 1908	Migraine.....1,500		0%	....	....
Buchanan: New York M. J. **113**: 45, 1920	Epilepsy.....	128	75%	....	....
Buchanan: Ibid	Epilepsy.....	..	....	10.5%	....
Buchanan: Ibid	Children from epileptic parentage...	202	16.9%	7.0%	....
Möbius: Die Migräne, Wien, A. Hölder, 1903	Migraine.....	..	....	90%	....

† Ascendants refer to the progentiors, or the familial antecedents, of the patient. Descendants concern the siblings, or the progeny, of the patient himself. Accordingly the percentages given should be interpreted with such consideration.

might coexist. Neusser [16] and Gansslen [17] discovered an eosinophilia in a high percentage of patients with migraine. Finally, Van Leeuwen

12. Pagniez, P., and Nast, A.: Essai d'une thérapeutique preventive de certaines migraines, Presse méd. **27**:172, 1919. Pagniez, P., and Nast, A.: Recherches sur la pathogénie de la crise de migraine, Presse méd. **28**:253 (April 28) 1920. Pagniez, P.: (Migraine) Presse méd. **29**:45 (Jan. 15) 1921.

13. Rohrer, F.: Anaphylaktische Erscheinungen im Symptomenbilde der Hemicranie. Ein Fall von Hemicrania ophthalmica, Med. Klin. **11**:862, 1915.

14. Kämmerer (footnote 1, reference 1).

15. Block, E. B.: Migraine, in Tice, System of Medicine, Hagerstown, Md., W. F. Prior Company, 1920, vol. 10, p. 3810.

16. Neusser, E.: Klinisch-lämatologische Mitteilungen, Wien. klin. Wchnschr. **5**:41, 1892.

17. Gansslen, M.: Die Eosinophilie bei der Migräne. Med. Klin. **17**:1232 (Oct. 9) 1921.

and Zeidner [18] isolated from the blood of patients with epilepsy, migraine, hay-fever and asthma a substance which constricts smooth muscle, and which was not found in the blood of normal persons.

Studies were conducted at this clinic to investigate the so-called common pathogenesis of allergy and migraine. In our series of 428 unselected cases, there were 36 of migraine, 38 of psychogenic headache (used as controls), 62 of allergy and 68 nonallergic patients (every fourth nonallergic patient in a group of 347 was chosen for

TABLE 3.—*Frequency of Migraine, Allergy or Both in Patient and His Family History*

Number of Cases Studied		Number and Type of Case		In Patient		In Family History		
				Allergy	Migraine	Migraine	Allergy	Migraine or Allergy or Both
I a	428	Migraine	36 8.4%	..	..	26 72.2%	15 47%	31 86.1%
b	428	Psychogenic headaches	38 8.9%	..	..	9 23.4%	7 18.4%	11 28.9%
II a	347*	Allergy	62 17.8%	..	9 14.5%	23 37%	24 38.7%	41 66.1%
b	347	Nonallergic (control)	68 19.5%	..	5 7.3%	9 13.2%	13 19.5%	24 35.2%
III a	347	Migraine	29	9 31.4%	..	..	..	..
b	347	Psychogenic headaches	22	4 18.1%	..	..	..	..
IV	347	Nonmigraine Nonallergic	201	..	..	39 14.4%	32 10.9%	58 28.8%

I b and III b controlled the migraine cases; II b controlled the allergic cases (the 68 cases represented every fourth nonallergic case in a series of 347 cases); IV controlled both series of migraine and allergy.

* Of the 428.

control purposes). Particular and specific inquiry was made concerning: (*a*) the familial distribution of migraine, (*b*) the familial distribution of allergy, (*c*) the familial distribution of either migraine or of allergy or of both in the patient who had either migraine or allergy and (*d*) the association of migraine and allergy in the patient who complained either of migraine or of allergy. The patients without knowledge concerning an adequate number of their blood relatives were excluded.

The foregoing controlled cases clearly portray the hereditary tendencies of migraine and of allergy, respectively (Ia and IIa). There was a family history of allergy in 47 per cent (Ia) of the patients with

18. Van Leeuwen, W. S., and Zeydner: Occurrence of Toxic Substance in Blood in Cases of Bronchial Asthma, Urticaria, Epilepsy and Migraine, Brit. J Exper. Path. **3**:282 (Dec.) 1922.

migraine, as opposed to 18.4 per cent (Ib)[19] in the patients with psychogenic headache, and 10.9 per cent (IV) in the nonallergic, nonmigrainous controls. There was a family history of migraine in 37 per cent (IIa) of the allergic patients as opposed to 13.2 per cent (IIb) in the nonallergic controls and 14.4 per cent (IV) in the nonallergic nonmigrainous controls. Further, 86.1 per cent (Ia) of the family histories of the patients with migraine revealed migraine, allergy or both, as opposed to 28.9 per cent (Ib) of the patients with psychogenic headache, 35.2 per cent (IIb) in the nonallergic controls and 28.8 per cent (IV) in the nonallergic nonmigrainous controls. Finally, 31.4 per cent (IIIa) of the patients with migraine were allergic as opposed to 18.1 per cent (IIIb) of the patients with psychogenic headache; 14.5 per cent (IIa) of the allergic patients were migrainous as opposed to 7.3 per cent (IIb) of the nonallergic controls; this figure closely approximates the figure for the prevalence of migraine.

The data on epilepsy are incomplete and confusing, and insufficient to warrant inclusion of such cases in this group. It is questionable whether heredity, per se, plays an important part in the incidence of epilepsy.[20]

SUMMARY AND CONCLUSIONS

1. A submerged intrinsic physiopathic mechanism is responsible for allergy and for migraine.

2. This mechanism is similar for both conditions, the clinical manifestations being regional expressions of the same process.

3. This mechanism is hereditary. Heredity is only partially selective in its regional expression; nonallergic persons with migraine have a higher percentage of allergy in their family history than do nonallergic persons who do not have migraine; likewise, nonmigrainous allergic persons show a higher percentage of migraine in their family history than do nonmigrainous persons without allergy.

4. Recent work by other investigators and my own studies on epilepsy would tend to preclude the possibility that epilepsy is due to the same mechanism that causes migraine and allergy.

---

19. Some of the patients with psychogenic headache were known to be allergic, which would account for the higher percentage of allergy in their family histories than was exhibited by the nonallergic, nonmigrainous controls.

20. Smith, N. R.: J. Michigan M. Soc. **24**:478, 1925. Hubbell, A. O.: Relations of So-Called Ophthalmic Migraine to Epilepsy, J. A. M. A. **51**:480 (Aug. 3) 1908. (footnote 10). Personal communication from Dr. Carl D. Camp, Ann Arbor.

Clinical and Occasional Notes

TORULA HISTOLYTICA MENINGO-ENCEPHALITIS*

GEORGE W. HALL, M.D., EDWIN F. HIRSCH, M.D.
AND HARRY MOCK, M.D., CHICAGO

Summaries of the knowledge concerning infections in man caused by *Torula histolytica* have been made from time to time since Stoddard and Cutler's[1] monograph was published; the most recent reports are those by Shapiro and Neal,[2] and by Rappaport and Kaplan.[3] In 1925, Shapiro and Neal, mentioned thirteen reports then recorded. They added another case and included a personal communication from Sheppe, thus making the total number of reported cases at that time fifteen, of which thirteen were infections of the central nervous system, one of the lungs and one of the muscles of the back. Rappaport and Kaplan reported a systemic torulosis and McKendree and Cornwall[4] in the same year reported an infection of the brain and meninges. An account by Hansmann[5] in 1924 of a case of meningitis and encephalitis caused by *Torula* is not mentioned by these investigators. Lynch and Rose[6] reported another case of *Torula* meningitis, and McGehee and Michelson[7] reported the case of a negress with a left inguinal abscess which contained *Torula histolytica.*

The purpose in this account is to record another case of *Torula* infection of the brain and meninges, with a statement of the clinical symptoms, the bacteriologic observations and the results of the postmortem examination. *Torula histolytica,* according to the present classification, is a yeastlike organism which reproduces by budding in tissues and in cultures, but which differs from the true yeasts by not fermenting sugars.

REPORT OF CASE

G. S., a white man, aged 33, married, entered St. Luke's Hospital, Chicago, on Nov. 4, 1926, complaining of severe headache of three weeks' duration,

---

* From the Henry Baird Favill Laboratory, St. Luke's Hospital, Chicago.

* Read at the Fifty-Third Annual Meeting of the American Neurological Association, Atlantic City, N. J., May, 1927.

1. Stoddard, J. L., and Cutler, E. C.: Monograph of Rockefeller Institute for Medical Research, Jan. 31, 1916, no. 6.

2. Shapiro, L. L., and Neal, J. B.: Torula Meningitis, Arch. Neurol. & Psychiat. **13**:174 (May 23) 1925.

3. Rappaport, B. Z., and Kaplan, Bertha: Generalized Torula Mycosis, Arch. Path. **1**:720 (April) 1926.

4. McKendree, C. A., and Cornwall, L. H.: Meningo-Encephalitis Due to Torula, Arch. Neurol. & Psychiat. **16**:167 (Aug.) 1926.

5. Hansmann, G. H.: Torula Infection in Man, Boston M. & S. J. **190**:917 (May) 1924.

6. Lynch, F. B., and Rose, E.: Torula Meningitis, Ann. Clin. Med. **4**:755 (March) 1926.

7. McGehee, J., and Michelson, T.: Torula Infection in Man, Surg. Gynec. Obst. **42**:803 (June) 1926.

tingling of the fingers for the same length of time and pain behind the eyes for twenty-four hours. He had consulted a physician just before the onset of the headache because of pain in the left arm and shoulder. He had received four injections of serum, and on the day following the last injection he complained of headache. This, he said, was constant and it seemed as though the head was compressed in a vise. He was unable to sleep because of the headache, except when relieved by acetyl salicylic acid or tablets for migraine. He had some difficulty in walking and vomited once. The personal and family histories did not contain anything noteworthy. As a child he had had measles. He used tobacco moderately, alcohol very seldom and he usually drank three or four cups of coffee daily. For the past few years, his time had been spent as a boy scout leader.

Clinical Findings.—The results of an examination of the eyes, by Dr. E. K. Findley, on Nov. 6, 1926, were reported as follows: "The pupils are moderately dilated, both optic disks are slightly edematous, and the vessels around them are tortuous. The edema of the optic disks is greater on the right side and measures about 1 diopter. It is not possible to determine whether there is a toxic neuritis or a slight papilledema from intracranial pressure." On this day, the patient complained of a sensation of pressure behind the right eye and a generalized headache. Diplopia, other cranial nerve paralyses, nystagmus, ataxia and paralysis of the upper extremities were not present. The results of tests for various reflexes were as follows: abdominal reflex, sluggish; left knee jerk, slight; achilles' reflex, active on both sides; Babinski and Oppenheim signs, absent; ankle clonus, absent; Kernig sign, on both sides doubtful.

On Nov. 7, 1926, there were: pain over the left eye; increased papilledema on both sides, that of the right disk measuring 2 diopters, that of the left less than 1; bilateral Kernig sign; soreness of the muscles of the neck, with anterior flexion. The leukocytes of the blood numbered 14,040 per cubic millimeter. A culture of the blood did not contain bacteria. On November 8, the edema of both optic disks had increased, and on November 9, there was choked disk on the right and a beginning choked disk on the left. The leukocyte count was 9,640; the blood pressure was systolic 118, diastolic 70; the temperature was 97.6 F.; the pulse rate 56 per minute, and rate of respiration 18. An exudate was expressed from each tonsil. Transillumination of the ethmoidal and maxillary sinuses, by Dr. L. Thompson, did not disclose any changes. Examination of the frontal sinuses was unsatisfactory.

Diagnosis.—An abscess of the brain, some form of meningitis, and possibly a brain tumor were considered as a clinical diagnosis. Because of the rapidly developing papilledema in the optic disks, decompression of the brain was regarded as a safer procedure than drainage by spinal puncture.

Operation.—On November 9, a right subtemporal decompression of the brain was made. When the dura was opened, a large quantity of cerebrospinal fluid escaped. No tumor or abscess was found by probing the brain with a dull needle.

Course.—November 10, there was ptosis of the right eyelid, and both optic disks were edematous. The next day there were: jacksonian twitching of the right arm, face, and leg; paresis of the left side of the body, and a positive Babinski sign on the left side. By this time, cultures of the cerebrospinal fluid, removed during the decompression operation, revealed opaque white colonies containing yeast organisms and the diagnosis of *Torula* meningitis was made.

The spinal fluid pressure, on November 12, was 300 mm.; on November 13, it was 340 mm. and after withdrawal of about 30 cc., the pressure fell to 120 mm. The patient became irrational on November 14; the face and chest were cyanotic and breathing was stertorous. The right arm and leg were in constant motion. The temperature rose to 103.6 F., and death occurred in the evening of Nov. 14, 1926.

Autopsy.—The postmortem examination was made on the morning of Nov. 15, 1926. The tissues of the trunk, head, and spinal canal were examined.

Anatomic Diagnosis.—The diagnosis was: acute *Torula* meningitis, marked edema of the cerebrum and of the meninges, multiple subpial hemorrhages of the brain, marked hyperemia of the leptomeninges of the brain and spinal cord,

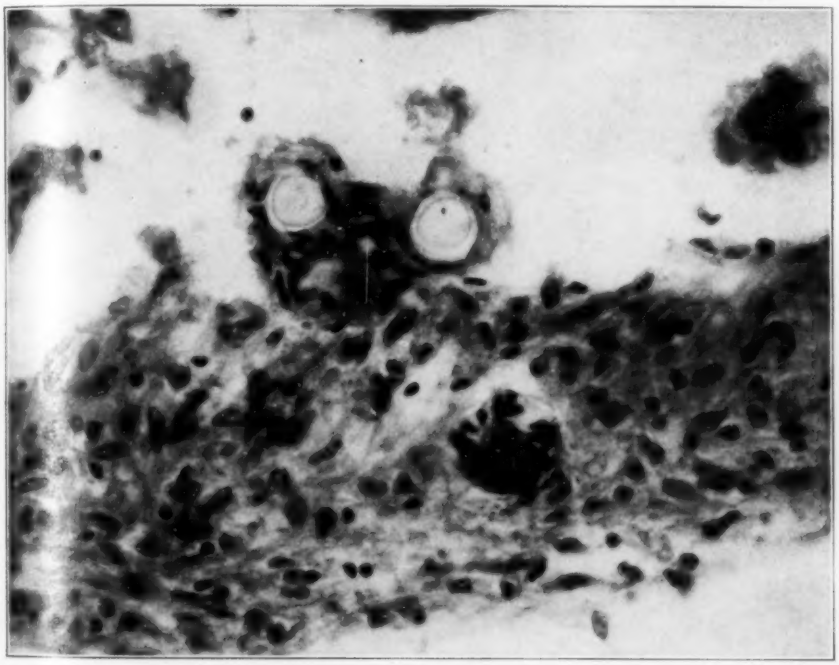


Fig. 1.—The tissue of the left gasserian ganglion containing torula organisms: × 375.

acute internal hydrocephalus, acute bilateral bronchopneumonia, bilateral fibrinous pleuritis, purulent tracheitis and bronchitis, recent unhealed trephine wound of the cranium, recent right subtemporal decompression wound, marked herniation of the cerebrum through the trephine hole of the cranium, passive hyperemia of the lungs and kidneys, cloudy swelling of the myocardium and parenchymatous organs, hyperplasia of the biliary and peribronchial lymph nodes, fatty changes of the liver, etc.

The brain (unfixed) with the top half of the dura weighed 1,460 Gm. There was marked distention of the leptomeninges by a faintly turbid, limpid and colorless fluid. In the pia-arachnoid were scattered thin, gray opacities from 2 to 3 mm. in diameter. Under the pia-arachnoid, in the brain substance,

were scattered hemorrhages, 1 cm. in their greatest diameter.[8] Gross changes in the middle ears, the sinuses of the dura and the cranium or in the gasserian ganglia were not noted.

Histologic Examination.—Sections from different parts of both lungs, chiefly the consolidated portions, contained regions of bronchopneumonia. Careful examination of many sections revealed no yeastlike structures. The dilated sinusoids of the lymph nodes in the hilum of each lung and the regional tissues contained masses of large mononuclear cells and leukocytes, but no yeastlike organisms. Other lymph nodes were without noteworthy changes. In the spleen were large recent hemorrhages and hyperplasia of the reticular cells. Slight fatty changes and passive hyperemia were the only changes of note

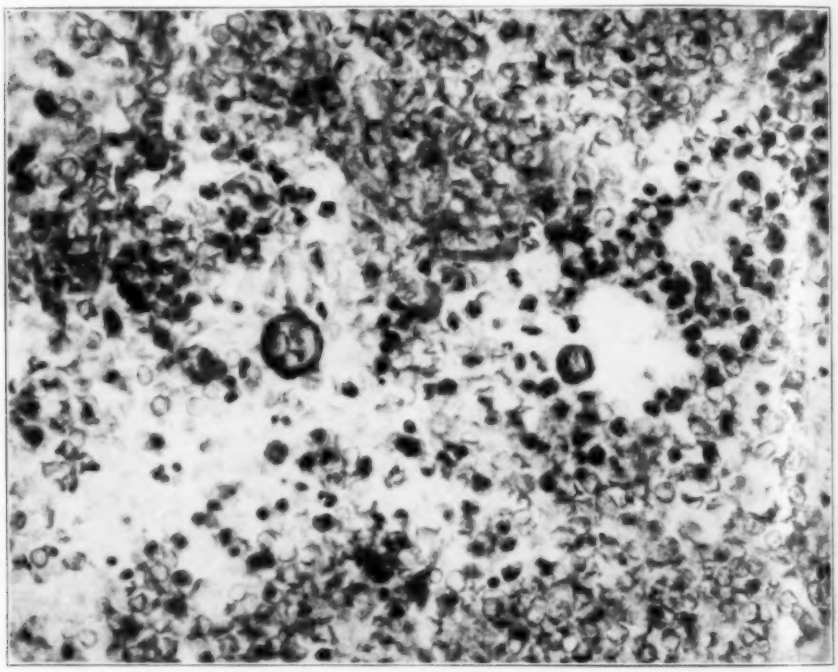


Fig. 2.—The torula organisms in traumatized brain tissue removed during the decompression operation; × 375.

in the liver. Tissues from the kidneys, aorta, urinary bladder, gallbladder, prostate, seminal vesicles, testes, vasa deferentia, suprarenal glands, and muscle of the heart were without noteworthy alterations.

Sections from each gasserian ganglion contained ganglion cells and nerve fibers, between which were ingrown bands of a loose fibrous connective tissue with many lymphocytes, practically no polymorphonuclear leukocytes, and with many yeastlike inclusions that varied in size between 10 and 19 microns. These organisms had been present sufficiently long to stimulate the fibrous and chronic granulation tissue reaction. Foreign-body giant cells occurred in the left gasserian ganglion. The hypophysis did not present any noteworthy changes, but in the adherent meninges were small masses of chronic granulation tissue containing yeastlike organisms.

8. A complete study of the brain and spinal cord is being made.

Bacteriologic Results.—Cultures of the cerebrospinal fluid taken during the decompression operation on Nov. 9, 1926, presented, after seventy-two hours in the aerobic containers, a heavy growth of round, slightly friable, cream-white, raised colonies with sharp edges. These, in stained preparations, were composed of yeast cells. Stains of the spinal fluid demonstrated the presence of yeast organisms. Cultures were made of the spinal fluid removed on Nov. 13, 1926, and twice on Nov. 14, 1926; they were examined in the same way, with identical results. A culture from the nose and throat made on Nov. 13, 1926, did not contain *Torula* organisms, nor were there organisms in cultures of the blood made on November 8 and 9. On March 20, 1927, cultures were made of spinal fluid removed on Nov. 14, 1926, and kept in the icebox; an abundant growth of *Torula* organisms was obtained.

Cultures of the fluids and tissues obtained at the postmortem examination were made with the following results: left lung, *Torula* not isolated; right lung, *Torula* not isolated; tracheal exudate, *Torula* not isolated; pericardial fluid, sterile; heart blood, pure culture of *Torula;* spleen, pure culture of *Torula;* spinal fluid, *Torula* isolated with *Streptococcus salivarius.*

Histologic examination of a small piece of tissue from the brain removed during the decompression operation contained torn substance of the cerebral

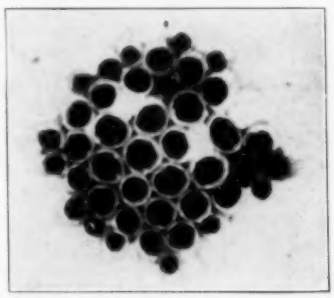


Fig. 3.—The torula organisms in cultures; × 1200.

cortex in which were yeastlike structures. Three strains of *Torula histolytica* obtained from: (1) spinal fluid during life, (2) heart blood (postmortem), and (3) spinal fluid (postmortem) did not ferment lactose, mannite, raffinose, salicin, levulose, maltose, xylose or adenite. Acid was formed slowly by the growth of these strains in dextrose and saccharose mediums.

COMMENT

Although reports of patients with *Torula* meningitis are relatively few, persistent severe headache is mentioned as a symptom in many of the accounts. Türk's[9] patient complained of severe headache and vomiting and had symptoms at first considered to be due to tuberculous meningitis. Evans,[10] Freeman and Weidmann,[11] Bettin,[12] Hansmann,[8] Shapiro and Neal,[2] McKendree and

9. Türk, Wilhelm: Deutsches Arch. f. klin. Med. **90**:335, 1907.

10. Evans, Newton: Torula Infection, California State J. Med. **20**:383 (Nov.) 1922.

11. Freeman, W., and Weidmann, F.: Cystic Blastomycosis of Cerebral Gray Matter Caused by Torula Histolytica, Arch. Neurol. & Psychiat. **9**:589 (May) 1923.

12. Bettin, Mona E.: Torula Infection, California & West. Med. **22**:98 (March 3) 1924.

Cornwall,[4] Rappaport and Kaplan,[8] and Lynch and Rose[11] mentioned this symptom in their reports. Other symptoms mentioned in these accounts include disturbances in vision, dizziness, ptosis of the eyelids and stiffness of the neck. All of these symptoms accompany meningitis, but, because of their chronicity and insidious onset, they resemble those caused by a tumor of the brain. The diagnosis of *Torula histolytica* meningitis is established by demonstrating *Torula* organisms in the spinal fluid.

How the organisms reach the meninges has not been determined. In the lungs of our patient, regions of chronic infection could not be considered the primary focus, and in microscopic preparations of these tissues, as well as in cultures, *Torula* organisms were not found. It seems, therefore, that the infection of the meninges in our patient came through the nasopharynx.

FRACTURE INTO THE FRONTAL SINUSES WITH DISCHARGE OF CEREBROSPINAL FLUID FOR OVER A YEAR

Report of a Case with Death from Pneumococcus Meningitis

ANDREW H. WOODS, M.D., PEKING, CHINA, AND
FRANK L. MELENEY, M.D., NEW YORK

Spontaneous discharge of cerebrospinal fluid through the nose without traumatic cause has been reported frequently.[1] An altogether different group comprises those cases of persistent flow of cerebrospinal fluid following fractures or operative procedures on the skull. In view of the frequency of fractures of the base of the skull and of the common textbook formula that cerebrospinal fluid may escape through such fractures, it seems remarkable that its occurrence is mentioned so infrequently in the copious literature on this subject describing specific cases. No doubt in many cases of fracture with a discharge of cerebrospinal fluid, the blood or secretions concealed it until death, or healing of the dural opening occurred promptly. Rawling,[2] in his Hunterian lecture on basal fractures of the skull, stated that in his cases 70 per cent of these fractures involved the sphenoidal sinus, and in many of these a probe could be passed through the nose into the cranial cavity. He also discussed in detail the various complications of fractures into the frontal sinuses and petrous bone, yet he referred to the outflow of the cerebrospinal fluid as a rare occurrence. In that lecture he stated that in 1840, Blaudin was the first to describe a case of flow of the subarachnoid fluid from the nose after a fracture. He refers to Goucard's and Malgaigne's cases of flow through the eustachian tube after fracture into the middle ear; also to the early case

1. Thomson, St. C.: Nasal Hydrorrhea, J. Laryngol. **13**:500, 1898. Glynn, T. R., and Glynn, E. E.: A Case Simulating Intracranial Tumour in Which Recovery was Associated with Persistent Cerebrospinal Rhinorrhea, Brit. M. J. **1**:871, 1905. McDougall, J. E.: Case of Idiopathic Cerebrospinal Rhinorrhea, Liverpool M. Chir. J. **30**:391, 1910. Locke, C. E., Jr.: Spontaneous Escape of Cerebrospinal Fluid Through the Nose; Its Occurrence with Brain Tumor, Arch. Neurol. & Psychiat. **15**:309 (March) 1926.

2. Rawling, L. B.: On Fracture of the Skull, Lancet **1**:973, 1034 and 1097, 1904.

of Van der Wiel (1727), and the later one of Langier (1839), in both of which the flow occurred through the external auditory meatus.

Archibald,[3] after reviewing the literature and his own experience in cases of fracture involving the base of the skull, considered that the escape of cerebrospinal fluid from the nose or ear is rare; it occurred in less than 7 per cent in one series quoted by him. Crandon and Wilson[4] found it in a little more than 5 per cent of 530 cases. Some cases have been reported of leakage through the nose following surgical operations on the base of the skull. One of the two otologic patients described by Cott[5] after an operation on the attic was troubled for some days with a flow of fluid from the external auditory meatus which appears to have been from the subarachnoid space.

Cushing[6] and Harmon Smith[7] referred to a patient of the latter's with a fracture of the floor of the frontal fossa, in whom a probe could be passed through the nose and between the frontal cerebral lobes. This patient had more or less continuous flow of cerebrospinal fluid from the nose and finally died of meningitis. The details as to the length of time the flow continued, the period after injury at which death occurred and the nature of the meningitis are not given.

The following case of a fracture of the skull connecting the subarachnoid space with both frontal sinuses was studied by us at the time of the accident, and the patient was observed for more than a year thereafter.

REPORT OF CASE

A French woman, aged 47, whose past history was irrelevant, ten or fifteen minutes before admission to the Peking Union Medical College Hospital, had been thrown from a rickshaw by collision with an automobile. She pitched headforemost to the ground and struck her forehead. She was unconscious for a few minutes, but answered questions when examined in the hospital. There was little shock and scarcely any disturbance in the function of the brain. The left eyebrow and the soft tissues of the frontal region presented a deep lacerated wound. Roentgenograms disclosed the extent of bony injury: The skull was fractured through both walls of the left frontal sinus and the roof of the orbit. Several fragments of bone had been driven through the dura and had lacerated the inferior surface of the left frontal lobe. There were two small tears on the anterior surface of the dura and one large tear running backward beneath the left frontal lobe over the orbit for an indeterminate distance.

Operation.—An immediate operation was performed by one of us (F. L. M.). The superficial wound was debrided, the loose fragments of bone were removed from the brain, the depressed roof of the orbit was raised, and the dura was sutured. It was not possible to close the long anteroposterior tear completely.

---

3. Archibald, E.: Surgical Affections and Wounds of the Head, in Bryant and Buck: American Practice of Surgery, New York, William Wood & Company, 1911, vol. 5, p. 75.

4. Crandon, L. R. G., and Wilson, L. T.: Fracture of Base of Skull, Ann. Surg. **44**:823, 1906.

5. Cott, G. F.: Cerebrospinal Fluid From the Ear, J. Laryng. Otol. & Rhin. **24**:74, 1909.

6. Cushing, H.: Surgery of the Head, in Keen: Surgery, Philadelphia, W. B. Saunders Company, 1908, vol. 3, p. 123.

7. Smith, H.: The Nose and Its Accessory Sinuses, in Keen: Surgery, 1908, vol. 3, p. 422.

Sutures were taken from before backward for about 3 cm., but at the posterior end a small opening was left. It was thought that drainage at this point for a short time might be advantageous. A small rubber drain was placed down to the dura. The deep periosteal fascial layer was closed about the drain and the skin sutured separately.

Course.—The drainage from the wound decreased rapidly, and the drain was removed on the fourth day. Healing took place without infection. There was no evidence of meningitis or of increased intracranial pressure. A mild postoperative pneumonia developed, but did not seriously interfere with recovery.

The left facial muscles were weak for the first ten days, and the right limbs were somewhat weak for a month. The left eighth nerve did not show any disturbance of function. There was bleeding from the external auditory meatus when the patient was first examined, but the cerebrospinal fluid did not escape. These signs were interpreted as indicating a fracture of the petrous bone with injury to the facial nerve and slight implication of the left pyramidal bundle in the pons.

Three weeks after the accident, the patient noticed a clear fluid dropping from her left nostril. This was marked when the head was erect or bent forward, but slight when the head was bent back or the patient was recumbent. Intranasal examination showed that this fluid came from the region of the left infundibulum. The drops formed in that position, pulsated for an instant, and then were dissipated over the nasal mucous membrane. At the time of the appearance of the nasal discharge there was a puffiness of the left malar region extending to the side of the nose. Pressure on this was followed by increased discharge from the nose.

The discharge of cerebrospinal fluid continued, with intermissions of a few days at a time, for at least thirteen months while she was under observation, and probably four or five months longer. The patient complained of slight headache and dizziness during the periods when the flow ceased, and of relief from these symptoms when the flow started again; but there were no signs at any time of serious rise in intracranial pressure. The optic disks, the blood pressure and the pulse rate were not disturbed. The lacrimal sac and duct were not involved in the fracture.

The patient was readmitted from time to time during the ensuing year for slight infections in the upper respiratory tract and other minor complaints, the next to the last time being thirteen months after the occurrence of the fracture. Each time the cerebrospinal leakage was observed, and the danger of ascending infection was considered. During this long period, the question was repeatedly brought up with regard to the wisdom of attempting to close the fistula by an operative procedure, but it was considered best to wait for spontaneous closure.

Eighteen months after the accident, the patient was suddenly overcome by an intense, fulminating cerebrospinal meningitis, of which she died within two days of its onset. On admission to the hospital, she was comatose. The spinal fluid was gray and had 9,400 cells per cubic millimeter. Glycosuria and hyperglycemia were present, although the urine had not shown any reducing substance at any time during previous admissions. Laboratory study of the spinal fluid showed the presence of pneumococcus group IV.

Necropsy.—Necropsy revealed a purulent leptomeningitis extending over the brain and spinal cord. A hole, 1 cm. in diameter, was found in the frontal bone above the left frontal sinus. The dura was adherent to the overlying

fascia. Below this there was a fault in the inner table of the anterior cranial fossa about 1 by 2 cm. in size, involving the vertical plate of the left frontal bone just above the orbit and extending somewhat to the right of the sagittal plane, opening into both the left and the right frontal sinuses. The septum between the sinuses was absent, and a defect in their floor at that point gave free communication with the nasal cavity. The membranes around the periphery of the bony opening appeared to be continuous with the mucous membrane of the sinuses. A part of the left frontal lobe of the brain had herniated somewhat through the opening, and a large mass occupied all of the left and part of the right frontal sinus. This mass on section proved to be made up of a small amount of brain tissue with a much larger amount of granulation tissue and partly organized coagulum and pus. The base of the brain was covered with a profuse purulent exudate. The pathologist's conclusion was as follows: "It is clear that the meningitis arose as a complication of the persisting fistula between the subarachnoid space and the frontal sinuses."

COMMENT

Long Continuance of the Cerebrospinal Fluid Leakage.—The first appearance of the leakage through the nose was three weeks after the accident; but, after the healing of the external wound, the accumulation of fluid beneath the soft tissues of the infraorbital region showed that escape of fluid was taking place during the first weeks through the fractured bone and around the mesial side of the orbit into the soft tissues. After that route was sealed, the fluid escaped through the nose.

The quantity of fluid lost by this patient was not determined, but in some of the cases of cerebrospinal fluid leakage, as much as 1.5 liters a day were measured (Giss' case quoted by Levinson[8]). McCormack's patient (cited by Rawling[2]) lost 3 pints in five hours.

The amount of fluid produced in patients, while abnormal discharge of the fluid continues, is not an index of the normal rate of its secretion. The whole truth as to the source of the cerebrospinal fluid and as to its ultimate escape from its rigid craniospinal encasement is still far from being known; but, under most circumstances, the rates of production and of escape are mutually adjusted so as to maintain a sufficient quantity of the fluid within the ependymal and subarachnoid cavities, and to maintain its pressure at a little above that of the venous blood of the intradural sinuses. The range within which this adjustment can be maintained under pathologic conditions is limited. In case of leakage, vital and mechanical devices for closing the abnormal opening are provided by the rapid healing of dural tears; or there is plugging of the bony opening by the membranes or even by the essential nervous tissues themselves. Chemical analysis would probably show that, in most cases (as reported by Pike[9]), when wastage occurs the quantity of fluid is kept up, even at the expense of quality.

After traumatic tears and punctures into the subarachnoid space, the defect in the membranes tends to heal spontaneously and quickly. Prompt healing is the rule almost without exception after gunshot and puncture wounds, and after fractures of the skull from blows. Cerebrospinal fluid leakage may cease

---

8. Levinson, A.: Cerebrospinal Fluid in Health and Disease, ed. 2, St. Louis, C. V. Mosby Company, 1910, p. 39.

9. Pike, N. H.: A Case of Cerebrospinal Rhinorrhea with Double Optic Atrophy, Brit. M. J. **1**:1104, 1910.

within a few hours or after one or two days. Rawling[2] considered a case of leakage for one month (Savory's) as worthy of special mention. We have observed cerebrospinal fluid cysts in the convexity of the cranium and over the spinal column following operative and other invasions of the subarachnoid space, but these cysts disappeared after from one to fourteen days. Even in the many cases in which surgeons have purposely sought to maintain cerebrospinal fluid drainage to relieve hydrocephalus, it has been difficult to prevent the rapid sealing-off of the subarachnoid space. Whether the artificial fistula was made to open on the skin surface, into a subaponeurotic layer, into the retroperitoneal tissues, into the peritoneal cavity, or into the venous sinuses or the extracranial veins, in the patients in whom infection did not take place the operation nevertheless nearly always failed because of the rapid closure of the dural opening.

Cerebral Herniation Into the Frontal Sinuses.—The development in this patient of an encephalocele through an opening into the frontal sinus is interesting and unique. We do not know of any other fracture in which it occurred.

There was no sign at any time of more than trifling headache or of other signs of intracranial pressure during the eighteen months after the fracture was received.

Increase in intracranial pressure is capable of causing herniation as is often seen after decompression for the relief of preexisting excess pressure. But when openings in the skull have resulted from accidents, often, as in our patient, there is no increase in pressure, but the herniation results from the fixation of a limited portion of the brain by adhesions to the periphery of the opening, after which inflammation and edema in that portion and in the tissues behind it cause the swelling mass to protrude. This process, as a rule, seals off the subarachnoid space and stops the discharge of cerebrospinal fluid. The fact that cerebrospinal fluid continued to flow in our patient may mean that the frontal sinuses were so nearly sterile for more than a year that adhesions, inflammation and herniation did not at once follow; but, on the advent of micro-organisms into the sinus, local meningitis, sealing off of the opening, herniation and then general meningitis occurred.

Terminal Infection.—One of the most interesting features of this case is the final penetration of virulent organisms into the physiologic interior of the body and the development there of a fatal infection. When fractures involve the nasal cavity, the chances of infection are greater than when the ear is involved. It is presumable that the meningeal infection in this patient took place through the nose, but it is not possible to say whether the infection at length reached the general pia-arachnoid by way of the fistula or indirectly through the protruding mass of membrane and brain. It seems probable that the outflowing of the fluid tends to wash away micro-organisms, and Rawling quotes limited statistics in support of that opinion. In that case organisms would not be likely to spread against the stream unless there were some obstruction to the drainage. The longer the tract the less danger there is for the organisms to reach the vulnerable spot. The nasal sinus mucous membranes and their secretion have certain defensive properties against micro-organisms. Probably infection in this case awaited the advent of a particularly virulent organism and a temporary blockage of discharge. This brings up the question of the soundness of our judgment when we decided not to attempt closure of the sinus by secondary operation during the course of the year and more throughout which the leakage was maintained.

EXCESSIVE MOVEMENT IN LAUGHING ON SIDE OF FACE PARALYZED FOR VOLUNTARY INNERVATION IN CENTRAL LESION OF FACIAL FIBERS

WILLIAM G. SPILLER, M.D., PHILADELPHIA
Professor of Neurology in the University of Pennsylvania

It has been known for many years that in certain cases of central facial palsy, the muscles on one side of the face which fail to contract to voluntary innervation may do so in laughing. Monrad-Krohn has shown that the contraction of the facial muscles in laughing may be even greater on the side paralyzed for voluntary innervation than on the normal side. At the meeting of the American Neurological Association in May, 1927, he reported cases of this type but did not give any explanation. It seems to me that the condition of the deep sensation of the facial muscles may have some influence over the degree of contraction of these muscles, and that when this sensation is lost, contraction of these muscles may be affected. I made reference to this fact in discussing Monrad-Krohn's paper.

In April 1906, I reported[1] two cases before the Philadelphia Neurological Society in which deep pressure sensation was preserved after removal of the gasserian ganglion for tumor of the ganglion,[2] and in one of these the sensory root of the fifth nerve was also divided behind the tumor. I do not know of similar observations antedating mine. A brief reference to these cases is made in the *Journal of Nervous and Mental Disease*. In lecturing to the students of the University of Pennsylvania Medical School, in 1907, I suggested that one or two of them might make this observation the subject of study; in this way, the paper by Ivy and Johnson was written and published in the *University of Pennsylvania Medical Bulletin*, in May 1907, from my service with my notes. In another paper,[2] I suggested that when deep pressure sensation was lost for a short time after a central operation on the fifth nerve, it might be explained by the swelling of the peripheral branches of the fifth nerve implicating branches of the seventh nerve with which the branches of the fifth nerve were in contact.

The observation that deep pressure sensation is conveyed in the seventh nerve has been confirmed by the work of Davis, Gerard (1923) and Souques, and by Hartmann in his thesis (1924) which confirmed the observation in thirty cases of retrogasserian neurotomy.

The recent work of O. Foerster has shown that the representation of sensation in the postcentral gyrus is somatotopic, as the representation of motion is somatotopic in the corresponding part of the precentral gyrus. As the motor fibers for the face are contained in the same nerve with the fibers of deep sensation, it is probable that the deep sensation of the facial nerve has a cortical and subcortical representation close to that of motion, probably closer than that for ordinary sensation of the facial supply. A lesion of the motor represensation above the reflex arc might, therefore, easily implicate deep sensation, partly or completely; complete loss of the latter might be the cause of excessive movement of the facial innervation, even greater than on the normal side, in laughing, which is a reflex movement and which in the cases under consideration is uncontrolled by the regulating function of deep sensation. I have made some personal observations which seem to indicate that deep sensation is represented in the postcentral gyrus.

---

1. Spiller, W. G.: J. Nerv. & Ment. Dis. **33**:736 (Nov.) 1906.
2. Spiller, W. G.: Am. J. M. Sc. **136**:712 (Nov.) 1908.

Davis, Souques and Hartmann have shown by electrical stimulation of the face that the recognition of position in the face is preserved after central operation on the fifth nerve and, therefore, probably depends on the integrity of the seventh nerve. It would be interesting, in such cases as Monrad-Krohn has described, if one could be found in which there was loss of function also of the fifth nerve, to determine whether the deep sensation is impaired or lost by the cortical or subcortical lesion; this could be determined satisfactorily only when loss of sensation of the fifth nerve had occurred, as otherwise it would be difficult to test deep sensation.

Since attention has been directed to these cases, they may be observed more frequently. It is probable that the case described by Herrmann under the title of homolateral mimic facial paralysis in frontal lobe lesion[3] really belongs to this group.

---

3. Herrmann: Monatschr. f. Psychiat. u. Neurol. **62**:52 (Sept.) 1926.

Abstracts from Current Literature

On the Mossy Fibers and Certain Doubtful Points of the Structure of the Cerebellar Cortex. S. R. Cajal, Trav. du labor. de recher. biol. de l'Univ. de Madrid **24**:215 (Dec.) 1926.

This is a critical study of several details of the structure of the cerebellum in man and other mammals. The technics used were: Bielschowsky's method and its modifications; the author's silver impregnation method after preservation in different fluids, and his latest impregnation methods for sections (pyridine-silver nitrate and alcohol; formaldehyde-silver nitrate and pyridine).

Cerebellar Glomeruli.—The aspect of the glomeruli (where the synapse between the dendrons of the grains and the branches of the mossy fibers takes place) varies considerably according to the silver impregnation method used, the species studied, age, amount of silver absorbed by the nerve arborizations and other undetermined factors. Three types of images are obtained:

1. Diffuse Nerve Plexus, Without Glomeruli or Conspicuous Arborizations of the Mossy Fibers and Grains. This is the usual appearance in sections of material preserved in mixtures of alcohol and ammonia, pyridine, chloral hydrate, and in sections stained with Bielschowsky's procedure. In such sections medullated axons, the recurring collaterals of the Purkinje cells, the climbing fibers, the baskets, etc., are the only stained structures. Occasionally a few branches of the mossy fibers appear impregnated.

2. Almost Exclusive Appearance Within the Glomeruli of Numerous Collateral and Terminal Arborizations of the Mossy Fibers. This image is obtained in sections stained with diverse neurofibrillar methods. According to the arrangement and extent of the branches of the mossy fibers two types of glomeruli can be distinguished: (a) large glomeruli, with extensive and complicated arborizations, and (b) middle and small sized glomeruli, with shorter branches, scantily divided and, sometimes, of fingerlike aspect. Large glomeruli usually contain terminal branches, while middle and small sized glomeruli generally enclose collateral arborizations.

3. Cerebellar Glomeruli in Which, Besides a Certain Granular Background, and Branches of the Mossy Fibers, There Is a Complicated Plexus of Fine Filaments and the Dendrons of the Granules. This image, the most complete and most difficult to analyze, is obtained through the use of one of the author's latest methods for sections (pyridine-silver nitrate and alcohol). Although human material, dog, cat, and other animals can be used, best results are obtained in young rabbits (from 3 to 4 months of age). The aspect of the sections strongly suggests, as already indicated by the author, that there is, besides the dendrons of the granules and the collateral and terminal projections of the mossy fibers, a third factor in the constitution of the glomeruli. This is represented by the last arborizations of the axons of the cells of Golgi. In addition there is a granular interstitial matter which fills the interstices of each intraglomerular plexus. This substance is stained after treatment with certain acid aniline dyes, also after the use of certain mordants (iron alum followed by silver impregnation, etc.).

The mossy fibers of the mouse have less complicated collateral and terminal arborizations than the corresponding structures of the rabbit. In certain sections the terminal arborizations of the axons of the cells of Golgi appear almost exclusively stained under the aspect of a delicate, compact plexus.

The uppermost arborization of the mossy fibers is placed in the immediate vicinity of the cells of Purkinje, usually in the spaces left between the baskets. This terminal arborization is not always associated with glomeruli, and when

associated the glomerulus exhibits a simple structure. Furthermore, Cajal has never been able to find any connection between the terminals under discussion and the dendrons of the granules or the axons of the cells of Golgi. The possible connection of mossy fibers with the soma of the cells of Golgi also seems doubtful, but the author remarks that this aspect of the problem cannot be solved in sections stained with neurofibrillar methods, and that only the use of Golgi's method will throw light on this debated point.

There occurs, in the layer of the granules, a peculiar type of cell, reported by Cajal and Illera in 1907, and well studied by Estable. Such cells are characterized by their connection with a compact plexus of branches arising in the baskets, but the behavior of their axon is not known. Since neurons with a short axon usually lack pericellular plexuses it is assumed that the cells under discussion have long axons entering the white matter.

The development of the neurofibrils in the arborizations of the mossy fibers is described. The branches begin to appear from the eighteenth to the twentieth day of postnatal life (in a cat, dog, or rabbit). At first they are rudimentary and do not possess neurofibrils and the latter do not appear until the granules and their dendrons have attained full development. In most of the cases the first neurofibrils arise in the end of the mossy fiber (anlage of the terminal arborization). The attraction leading to the formation of the synapse between the dendrons of the granules and the mossy fibers seems to be conditioned by the neuroplasm rather than by the development of the neurofibrils.

The finer structure of the cerebellar grains is also described in detail.

Terminal Baskets ("Endkörben" of Kölliker).—The section of the paper devoted to these structures is not descriptive but the author emphasizes some sources of error insufficiently considered by the defenders of the theory of the continuity of the neurons. From this analysis he concludes that his earlier views on the absolute independence of the arborizations of the baskets and the soma of the Purkinje cells are still supported by the facts. In the light of his recent work on the subject he states that the aspect and complication of the baskets vary according to the animal and its stage of postfetal development. The baskets become more and more complex with age. Contrary to almost universal belief, the architecture of the cerebellum does not attain all of its perfection with the acquisition of the faculties of locomotion and equilibrium, but continues its progressive development until the animal has reached the adult stage and has attained all its habits and higher instincts.

Some Problems on the Structure of the Plexiform Layer.—Besides the superficial and deep stellate cells, climbing fibers, parallel and transverse fibers, etc. of the plexiform layer there are two types of fibers of uncertain origin and connections. These are called by Cajal "deep parallel fibers" and "long or radial medullated tubes," respectively.

Deep Parallel Fibers: These fibers were observed by the author in 1903 in sections stained with neurofibrillar methods, where they appeared deeply stained. This peculiarity was regarded as caused by their deep position. More recent studies by Schültze, and Stöhr with other neurofibrillar methods, have shown that the fibers under discussion always appear deeply stained. Stöhr seems to regard them as a new factor in the structure of the cerebellum and has termed them "fibers of Schültze."

According to Cajal the deep parallel fibers are the continuation of the axons of the granules; they differ from other parallel fibers of similar origin in that they attract more of the silver stain. He suggests that the difference in staining of the parallel fibers is probably due to the fact that some of these elements contain thick axons. It seems likely that the latter arise in certain moderately large grains which in the cat and other adult animals appear sporadically stained by the neurofibrillar methods. This interpretation is to some extent supported by the fact that such neurofibrillar methods as

those of Bielschowsky, Liesegang, Schültze, and Cajal's own methods seldom stain very thin amedullated axons.

Long or Radial Medullated Tubes: These are straight fibers arising from the granular layer and gradually disappearing toward the upper half of the plexiform layer. They are readily seen in sections stained for myelin (Weigert, Spielmeyer, etc.). The origin and mode of termination of the radial medullated tubes could not be solved in sections stained with the neurofibrillar procedures, where they appear occasionally impregnated. Possibly these tubes are long recurring collaterals of the axons of the Purkinje cells, ending in the terminal arborizations of these elements; if this is true they would constitute an association system between Purkinje cells located in wide areas. But the final solution of the problem rests on a study of sections stained with the method of Golgi.

Morphologic Evolution of the Arborization of the Purkinje Cells.—Three phases are distinguished in the development of the Purkinje cells: (a) spindle-shaped corpuscles; (b) stellate cells with dendrons lacking definite orientation, and (c) stage of orientation and flattening of the arborization.

Spindle-Shaped Corpuscles; The neurofibrils of the Purkinje cells do not attract the silver stain in chick embryos until the twelfth or thirteenth day of incubation. More constant impregnations are obtained during the fourteenth day. The cells appear at this age as spindle-shaped corpuscles arranged in several layers. The axon arises in the lower end of each cell, while the upper end is continued into a few dendrons, which appear as bundles of neurofibrils scantily branched. The nucleus is oval and is usually placed in the lower half of the cell. In mammals, specially in the albino mouse, spindle-shaped Purkinje cells are found only during the last days of fetal life (vermis). In the flocculus such corpuscles still occur in the new-born. As in the chick embryo they are arranged in several layers.

Stellate Cells with Dendrons Lacking Definite Orientation: During the second phase great numbers of lateral, ascending dendrons are produced. The dendrons are branched and end as conical projections. Tangential sections of the cerebellar lamellae show that the arborization of the developing cells is not flattened. In newly hatched birds (swallow, sparrow, etc.) the Purkinje cells, as shown by Tello in heron embryos, do not appear as a continuous layer but form groups separated by pale zones or bands oriented in an antero-posterior direction. In the newly hatched swallow there are eight or more of these groups in each lamella. The groups are recognizable in the fourteenth day chick embryo.

Afferent fibers begin to appear during this stage.

Stage of Orientation and Flattening of the Arborization: This stage is characterized by the following features: (a) formation of granular or plexiform layer at the level of the dendritic arborizations; (b) successive flattening of the latter associated with the production of more secondary and terminal branches; (c) disappearance of the lateral and descending dendrons arising from the soma, and (d) increasing differentiation in the area of the superficial grains of two layers, namely, a lower layer of spindle-shaped, longitudinal elements (parallel to the cerebellar lamellae), and an upper layer made up of polygonal cells some of which show signs of mitosis. The latter layer represents the germinal or indifferent zone that later will produce the grains and neurons of the adult plexiform layer.

The degree of development of the dendritic arborization increases with age, in birds as well as in mammals. Neurofibrils are well developed at the beginning of this stage and the axons appear deeply stained in the slides. Some unusual details, of difficult interpretation, occurred in the preparations. Thus, it is frequent to find in the cerebellum of puppies (4 to 8 days of age) numerous displaced fibers with clublike endings; some thin and others thick. The nature of these fibers is uncertain, but the author suggests that they are arrested mossy fibers. Other fibers arise in the layer of the granules, travel

through the molecular layer and bifurcate in the zone of horizontal cells. This is interpreted as precocious formation of the axons of the horizontal spindle-shaped cells, and furnishes a new example of aberrant migration and development of the embryonic neurons. These abnormalities are more frequent in the cerebellum than in other parts of the central nervous system.

Another peculiar feature in the Purkinje cells of young animals, already reported by Tello in newly-hatched birds, is a scarcity of the neurofibrils in the reticulum; those neurofibrils that are present show spindle-shaped thickenings forming a coarse network. Similar images occur in young animals subjected to cold, and it seems likely that the cause of the phenomenon is due to cooling of the eggs of the wild birds studied (sparrow, swallow, magpie, etc.). The same effect may be experimentally produced through cooling of hen eggs for a few hours before removal of the embryo.

The paper ends with a brief discussion of the factors concerned in the differentiation of the arborization of the Purkinje cells.

NONIDEZ, New York.

COUNTERPULL (MOTOR NEGATIVISM), COMPULSIVE GRASPING AND THE OPTIC THALAMUS. K. KLEIST, Monatschr. f. Psychiat. u. Neurol. **65**:317 (June) 1927.

This article is an exhaustive discussion of a phenomenon for which Kleist has coined the name "Gegenhalten," which I have translated as "counterpull." Counterpull is a tonic contraction, manifest or latent, which can be brought out by passive movement of various parts of the body; it is the resistance with which these passive movements are met. The resistance of the patient increases with the force used by the examiner. The reaction is described in detail; it is brought into relation with other somewhat similar phenomena, such as the grasp reflex and the persistence of muscular contraction (as seen, for example, in myotonia). The author considers it a motor negativism, an affective disturbance, and describes its relationship to the negativism of the psychoses. He reports ten cases in which counterpull occurred as a focal symptom on the basis of cerebral arteriosclerosis. All showed bilateral thalamic lesions. He believes the rôle of the thalamus in these cases is inhibitory — disease of the thalamus releasing a lower affective center in the caudal oblongata, which is the actual reflex center for counterpull.

The author's summary is: 1. The pure motor manifestations of negativism are termed "counterpull." By this he means that when the examiner attempts to change the position of part of the body, he is met with a resistance — a muscle tension — which increases in proportion to the force used by the examiner, and which tends to maintain the part in the original position.

2. Counterpull can be local — confined to certain parts of the body. It has certain predilection zones: the elevators of the lower jaw, muscles of the neck, muscles of the trunk and proximal limb segments and the adductors of the arms and legs. Local counterpull is commonest in the lower jaw. A resistant (negativistic) personality reaction is often lacking.

3. Counterpull can often be brought out in muscles which do not show it at first, by repeated, slight stretching.

4. Counterpull may be either reactive (directed against passive change of position) or spontaneous (present even when external force is not applied).

5. Counterpull plus akinesia may lead to a peculiar type of motor incapacity.

6. Counterpull is a compulsive resistance and at times is associated with peculiar feeling tones. It belongs in the larger field of the affective disorders.

7. Counterpull occurs not only in catatonia, but also in other psychoses, especially in those in which psychomotor disturbances are common. But it is also found in focal diseases of the brain, such as softenings from arteriosclerosis, hemorrhages and tumor.

8. In general, counterpull as seen in focal disease is not so severe as in the psychoses; furthermore, when it occurs in focal disease, it is accompanied by other focal symptoms, which give a picture different from that seen in the psychoses.

9. Slight hemiparesis and pyramidal symptoms increase counterpull on the affected side, or render a latent counterpull manifest on that side; more marked pareses and paralyses wipe it out, so that in severe bilateral disease of the pyramidal tract, it may be evident only on the less paretic side.

10. When counterpull occurs as a result of focal disease, it is often associated with a grasp reflex of the hand, less often a sucking reflex of the lips.

11. There is probably a form of firm clamping grasp reaction, produced by a strong, grasp reflex, constantly renewed by an attempt to withdraw the hand. This is the compulsive grasp (Zwangsgreifen) in the narrower sense. In contrast to counterpull, it is confined to the hand and cannot be produced at will.

12. Persistence of contraction (Kontractionsnachdauer) also occurs at times with counterpull. Like counterpull, it is not confined to the hand. But it differs from counterpull in that it cannot be produced at will, that it passes off spontaneously after a time, and that the force of the contraction does not increase with the resistance. Persistence of contraction is related to rigor, and to the tonic innervations in athetosis and torsions, and often accompanies them. On the other hand, persistence of contraction differs from the grasp reflex in that it is not the result of a reflex contraction, but of a voluntary one.

13. Ten cases of cerebral arteriosclerosis are reported, in all of which the phenomenon of counterpull was shown. Nine of them showed the grasp reflex. All had bilateral larger or smaller foci of softening and lacunae in the thalamus. In most of the cases disease of the thalamus was evidenced by other affective disturbances as well (for example, compulsive crying).

14. Associated disease of the striatum produced tremors and iterations; in a few cases associated pallidal disease caused rigor or persistence of contraction. In three cases there were choreatic or athetotic movements or alternating motor restlessness, probably due to the thalamic or subthalamic foci, possibly with involvement of the putamen.

15. As to the hemispheres, the only uniform lesions were in the motor cortex and its corona, the internal capsule and pes; this involvement was relatively slight, usually bilateral, sometimes unilateral. The distribution of the counterpull was determined by these pyramidal lesions.

16. Compulsive counterpull, released by disease of the thalamus — the higher affective center — is probably developed in a lower affective zone, in the association field of the caudal oblongata; the grasp reflex is probably developed still lower, in the cervical cord. In favor of this view are observations in anencephalic cases (oblongata and midbrain cases) and cases of hemorrhage into ventricles and into the brain stem, in which the grasp reflex, with or without counterpull, was retained.

SELLING, Portland, Ore.

THE QUESTION OF ACUTE TRAUMATIC SEROUS MENINGITIS. W. J. MATSCHAU, Ztschr. f. d. ges. Neurol. u. Psychiat. **108**:570 (May) 1927.

In 1869, Billroth introduced the term serous meningitis. Many questions regarding this still remain unanswered, among them the question of etiology. Some contend that serous meningitis occurs only secondary to trauma, others that it occurs in many forms of nervous diseases and with other forms of meningitis; and still others claim the occurrence of an idiopathic serous meningitis. In 1891, Quincke first established the exact diagnosis of serous meningitis, together with an accurate picture of the disease. In 1897, Boenninghans collected reports of twenty-eight cases from the literature in which twenty-one patients died and seven were operated on. In twelve of these cases the disease

in itself was not an entity, but occurred late in the disease as a final event before death. Boenninghans established acute serous meningitis as a disease entity. Others have reported it secondary to various infectious diseases such as influenza, typhus, pneumonia, measles and pertussis, and not infrequently after an acute otitis. In twenty-eight cases, Lewschin found it following otorrhea in six cases, and after pyemia in eleven cases. There are cases, however, in which acute serous meningitis occurs in a healthy person. Lewandowsky calls this the idiopathic type of serous meningitis. Quincke divides the disease into meningitis serosa externa, in which the exudate is subarachnoid, and meningitis serosa interna, in which the exudate is in the cerebral ventricles. The latter form he believed was due to an angioneurotic cause.

Acute serous meningitis develops as a complication in many infectious diseases. It also arises primarily, always with fever, and thus is hard to differentiate from an infectious disease. These facts tend to demonstrate that acute serous meningitis is a parasitic or infectious disease. Two points seem to contradict this view: (1) the absolute sterility of the fluid in acute serous meningitis; (2) the development of idiopathic serous meningitides after traumas of the head. With regard to the first point, the sterility of the fluid does not militate against an infectious origin. Many organisms produce distant effects by means of endotoxins or exotoxins, and the production of serous meningitis is due to the action of toxins produced by organisms elsewhere in the body of the animal. Why a toxin acting in this way should affect only the meninges is not explained by this theory. The occurrence also of serous meningitis after head trauma does not argue against the parasitic origin of the meningitis. Many diseases of the nervous system of definitely parasitic nature develop after trauma. Examples of this are tuberculous spondylitis, abscess of the brain, etc. Matschau says the organisms had already gained entrance to the particular tissue involved and the trauma set off a latent infection. Riebold believes acute serous meningitis develops from toxins without parasitic origin — as in obstipation and menses.

Quincke divided serous meningitis into: (1) meningitis serosa externa; (2) meningitis serosa interna. Boenninghans classifies them as: (1) meningoencephalitis serosa externa; (2) meningitis serosa interna. The pathologic changes in meningitis serosa externa are: severe hyperemia, edema, and local extravasations in the meninges. The subarachnoid exudate is transparent with a flocculent sediment. In meningitis serosa interna a flattening of the convolutions may be present. The pia mater of the lateral ventricles is edematous and infiltrated with round cells. The ventricles are dilated and filled with transparent or somewhat cloudy fluid. Hausemann found an increase in the connective tissue and endothelial cells in the pia mater. Steffen and Hergerenin found perivascular infiltrations in the pia in secondary serous meningitides of children. The cerebrospinal fluid is sterile. Riebold found a hemorrhagic serous exudate once. Finkelnberg has reported *Bacillus influenzae*, pneumococci, streptococci and staphylococci, in fluids, but no one has corroborated his observations.

Clinically, the disease begins with severe headache, with later vomiting and severe constipation. Simple optic neuritis is an early occurrence in the disease (neuritis optica simplex et edematosa). Fever then sets in and may reach as high as 39 C. (102.2 F.). The pulse rate may be slow or rapid; the respiration is superficial and slow, and sleep is disturbed. Consciousness is sometimes preserved, sometimes lost; even coma occurs. With the further course of the disease there occur severe pains in the upper and lower extremities, stiff neck, twitchings in the cerebral nerve distributions and in muscle groups in the extremities. In some cases the entire body may be involved. Pareses and paralyses may set in, and a positive Kernig sign may be found. The reflexes may be decreased or increased and at times a Babinski sign may be present. The amount of spinal fluid is greatly increased. It is under increased pressure and may vary from 150 to 600 mm. of water. The

pupils are irregular and even lose their light reaction later in the disease. The fluid is clear, has a low specific gravity, shows no organisms, and may show a slight increase in either lymphocytes or polymorphonuclear cells.

Matschau reports a case of acute serous meningitis which developed after trauma; the patient recovered. Patients are often incapacitated after the disease. They may grow progressively worse and die, or may develop optic atrophy or a chronic form of the disease with localized accumulations of fluid simulating a tumor of the brain. Many such cases have been reported. The only treatment of any value is repeated lumbar puncture.

ALPERS, Philadelphia.

GLOSSOPHARYNGEAL NEURALGIA (TIC DOULOUREUX). WALTER E. DANDY, Arch. Surg. **15**:198 (Aug.) 1927.

Dandy describes two cases of glossopharyngeal neuralgia and outlines an operative procedure for the relief of the pain. Clinically, glossopharyngeal neuralgia closely resembles major trigeminal neuralgia in the character of the pain. The distribution of the pain is the chief means by which the two conditions may be differentiated. The paroxysms of pain always strike some part of the glossopharyngeal sensory area — the tonsil, back of the tongue or the pharynx; the pain is always unilateral and induced by liquids or solids passing over the sensory endings of the ninth nerve in the mouth or pharynx. The pain is always excruciating; it is variously described as lancinating, knife-like or like stabs of a red-hot iron. Each individual pain usually lasts only a few seconds, rarely over a minute; the duration of the paroxysm, is, therefore, usually less than that of a trigeminal neuralgia, but seems more severe, if possible. There is a strong tendency for the paroxysms to occur in a series over a period of time varying from a few days to a few weeks; in nearly every case intermissions of several months have occurred. In nearly all cases pain radiates to the ear — the meatus, the concha, the lobule or in front of or behind the ear — and not infrequently it begins there, either before the more central pain, or almost simultaneously with it. Rarely does the pain radiate elsewhere than to the ear or its environs and to the affected side of the throat. Although the method par excellence of starting attacks of glossopharyngeal neuralgia is to drink hot or cold liquids, the attacks are also induced in many other ways. Other recognized causes are: swallowing, talking, yawning, coughing, shouting, sneezing, touching the ear, suddenly turning the head or touching the angle of the jaw. In many instances the attacks seem to occur spontaneously. A trigger zone is often present, in the base of the tongue near the tonsil, the pharyngeal wall or the tonsillar pillar. Since the type of pain in glossopharyngeal neuralgia is very similar to that of trigeminal neuralgia, the distribution of the pain must be most carefully considered to make a proper differential diagnosis. For if the two conditions are confused and if treatment is directed against the trigeminal nerve when the glossopharyngeal is involved relief will not follow.

For the treatment of glossopharyngeal neuralgia Dandy advocates unilateral suboccipital craniectomy with section of the nerve intracranially in the posterior fossa. This procedure affords adequate exposure of the nerve which may be easily identified, isolated and sectioned. Cutting the nerve in this locality precludes the possibility of its regeneration and the recurrence of the pain. Postoperatively, the results have been excellent, complete relief following section of the nerve.

Following glossopharyngeal neurectomy motor impairment of any kind could not be noted on careful examination nor did the patient complain of any difficulty in swallowing. No disturbances of salivary secretion could be determined. The sensory changes noted were complete loss of taste on the posterior two thirds of the tongue on the affected side and in one case some slight alteration in taste on the anterior two thirds. Anesthesia to touch

extended over the ipsilateral half of the uvula and over a narrow rim on the oral surface of the soft palate and over a great extent on its nasal surface. A cap of sensation remained at the vault of the nasopharynx, doubtless being supplied by the trigeminal nerve. The remainder of the pharynx, anterior, posterior and lateral, down to the epiglottis (including the posterior aspect) the vallecula and the pyriform sinus, was anesthetic. Anteriorly and posteriorly the anesthesia ended sharply at the midline. The tonsil and eustachian tube were insensitive in both cases. The epiglottis was anesthetic on its posterior aspect; the line of demarcation of the area with the normal sensation of the anterior surface from that supplied by the vagus is a sharp line along the rim of the epiglottis. The precise line of demarcation between the area of normal and lost sensation was difficult to determine in two places — above at the vault of the nasopharynx and below at the beginning of the esophagus. The anterior wall of the sphenoid and the posterior end of both the middle and inferior turbinates were insensitive. There was no area of anesthesia in the nasal cavity. It could not be determined whether there was any loss of sensation along the posterior border of the vomer.

Dandy's communication is important for two reasons: (1) It goes far to confirm the investigations of others and to help to clarify many somewhat doubtful points concerning the sensory field of the glossopharyngeal nerve. Since the section of this nerve creates an isolated glossopharyngeal lesion, the sensory changes produced must be attributed to this nerve alone. (2) He adds a conclusive bit of evidence that glossopharyngeal neuralgia does exist as a separate clinical entity and that pain from this source may be promptly and safely relieved by section of this nerve.

GRANT, Philadelphia.

LESIONS OF THE MOTOR PLATES IN LEAD POISONING. J. M. DE VILLAVERDE, Trav. du labor de Recher. biol. d l'Univ. de Madrid **24**:267 (Dec.) 1926.

Daily doses of 1 cc. of 1 in 100 and 1 in 500 solutions of lead acetate were injected under the skin of the back of rabbits, cats and guinea-pigs. After a few days the animals began to grow thinner (particularly the cats) and showed symptoms of difficulty of locomotion, specially with the hind legs which tended to remain motionless. Some animals were killed before the onset of the symptoms, others were injected after severe symptoms had appeared.

Pieces of tongue, gastrocnemius, and soleus muscles were preserved in alcohol-ammonia, pyridine, and 10 per cent of liquor formaldehydi. Cajal's reduced silver nitrate and Boeke's modification of Bielschowsky's method were used for staining. Other pieces of muscle were fixed according to the technic devised by Heidenhain for the study of diverse details of the muscle fibers. Muscles from normal animals were also used as controls.

When poisoning is not too severe the nerve fiber reaching the plate usually has a slightly varicose aspect. The same occurs in that portion of the fiber within the motor plate. There are fewer branches than in normal plates. This scarcity is due to degeneration of some of the branches; their remnants appear as short conical stumps which in some instances consist of condensed argentophil material, in others the latter appears fragmented in small blocks. Atrophy of the branches may be widespread and in such a case only the main stem, continuation of the nerve fiber, persists, and it appears beset with conical projections, remnants of the secondary branches.

The process of degeneration and atrophy of the thin branches of the motor plate differs widely from the phenomena observed during wallerian degeneration. The neurofibrillar network of the arborization begins to disappear and the individual neurofibrils are completely destroyed. The argentophil material appears in some cases as if it were condensed around vacuoles, and it gives the impression of being agglutinated into fairly thick filaments. The branches show a double process of degeneration manifested in accumulation of the argentophil material in certain points, and its disappearance from

others; as a result of these phenomena the neuroplasm appears occupied by scattered masses of argentophil material. Sometimes the masses are exceedingly small. The origin of the varicose swellings of the branches is difficult to explain.

Another fact that deserves mention is that atrophy of certain small branches of the plate takes place through separation of the argentophil material from the neuroplasm. Swelling and the characteristic granular arrangement seen in the first stages of wallerian degeneration were never observed in the plates of poisoned animals.

The network of Boeke appeared stained in some fibers. In some cases it showed a granular aspect, the beginning of its degeneration. Evidence of the continuity of the trabeculae of the network with the argentophil material is not noted, a fact already reported by Cajal.

In cases of severe poisoning (guinea-pigs injected for several days after the onset of poisoning symptoms) the disturbances caused by the injections are more profound. The muscle fibers lose their cross striations; only the longitudinal striations are visible. Boeke's network does not stain. In other respects the structure of the motor plate has not changed, but the nuclei are somewhat smaller than usual, and their outline is less sharp than in normal plates. The nerve fiber reaching the plate appears moderately varicose; the varicose swellings are small and their arrangement is such that the fiber shows a corkscrew like appearance. In many plates the terminal arborization has been completely destroyed; others show remnants represented by deeply stained blocks of argentophil material not connected by neuroplasm as in cases of less severe poisoning. In many instances the plates still show some of the terminal branches. Separation of the argentophil material from the neuroplasm does not occur, nor vacuolization in those branches still present. The latter consist of deeply stained argentophil material with varicose aspect.

NONIDEZ, New York.

A CASE OF "SYMPTOMATIC" NARCOLEPSY. JULIUS ZADOR, Monatschr. f. Psychiat. u. Neurol. **66**:13 (Sept.) 1927.

CASE REPORT.—The patient was a married woman, aged 32; her father's family was long lived; the mother had epilepsy, the attacks being rather atypical. The patient when a child showed abnormal development of hair. During her school years she had attacks of vertigo or of unconsciousness. From the age of from 15 to 18 she had migraine. With the onset of menstruation the migraine stopped, but attacks of nausea and vomiting accompanied the periods. In 1921, a left oophorectomy was performed. Several months later, "sleep attacks" with loss of tone occurred. The attacks usually followed excitement, but occasionally came on without demonstrable cause. The attack itself was preceded by a sense of nausea, like an aura. She then became pale; this was followed by loss of tone, and then by sleep. The breathing was stertorous at first, then superficial. There was not any incontinence, or biting of the tongue, and the pupils reacted to light. During the first phase of the attack, she could not be aroused. Ordinarily, the attack lasted from two to three minutes. After it was over the patient could recall only the initial nausea. The attacks could be artificially induced by certain stimuli (music, odors, fright, etc.). Therapeutically, phenobarbital and bromide were without effect. The results of a physical examination were essentially negative. The patient was of a sensitive type with a labile pulse and a male type of distribution of hair. There was nothing suggestive of hysteria.

The author classifies the case as symptomatic narcolepsy, but with reservations. The inability to wake the patient in the early part of the attack suggests epilepsy rather than narcolepsy, and the mother's history is suggestive in the same sense. On the other hand, the spontaneous remissions, the refractory

action toward phenobarbital and bromide, the ability to produce attacks artificially by external stimuli, and the intimate relation of the attacks to excitement speak against epilepsy.

In general, in the literature, it is maintained that narcolepsy and epilepsy have nothing in common. But the author does not accept this point of view. Epilepsy is a reaction which can be brought about by stimuli arising in different parts of the brain — that is, it has to do with a reaction type and not with a localization. Narcolepsy, on the other hand, is a syndrome with a definite localization. One can conceive the process producing an epilepsy to be localized in the narcoleptic zone, "in the region of the posterior wall of the third ventricle and the centers in the adjacent gray of the interpeduncular region." In such a case the resulting narcoleptic attacks would be a subgroup of epilepsy. From the etiologic standpoint the justification for separation is no better. Both epilepsy and narcolepsy can be caused by various processes. Neither is an etiologic unit.

As to the significance of exogenous factors capable of producing attacks in the foregoing case (music, odors, fright), the author thinks they all act on the vasomotor center as does the psychic upset causing the spontaneous attack. The history indicates a marked lability of that center (syncopal attacks, migraine, angiospastic attacks, etc.).

From the author's standpoint, the essential significance of this case is that it presents some features suggesting narcolepsy and others suggesting epilepsy. He believes that one cannot draw an absolute line between the two conditions; and he thinks further that as more and more atypical cases are described, not only will the distinction between epilepsy and narcolepsy fall, but also that between the two groups of narcolepsy, idiopathic and symptomatic.

SELLING, Portland, Ore.

MALARIA TREATMENT OF CENTRAL NERVOUS SYSTEM SYPHILIS. PRELIMINARY OBSERVATIONS. J. R. DRIVER, JOHN A. GAMMEL and L. J. KARNOSH. J. A. M. A. **87**:1821 (Nov. 27) 1926.

Before reporting their own observations the authors present a historical review of the literature with a table of all cases of general paralysis treated with malaria that had been recorded up to April 1, 1926. Their observations are based on the reactions of seventy-nine patients treated between May 19, 1925, and March 15, 1926. Each of the patients received the same asexual strain of *Plasmodium vivax* from a case of naturally acquired malaria in the medical service. The technic is described in detail; in general, intravenous injection of from 2 to 8 cc. of blood was the method of inoculation. From twelve to sixteen chills were allowed, and termination was effected by the oral administration of 10 grain (0.65 Gm.) doses of quinine sulphate three times daily for three days and then 5 grain (0.32 Gm.) doses three times daily for four days.

Complications are next discussed and the following indications for interrupting the malaria treatment are given: (1) urea increase up to 70 mg. or over; (2) systolic blood pressure below 95 mm. between chills; (3) severe icterus with index above 50; (4) red cell count below 2,000,000; (5) rapid anemia with cessation of chills; (6) profuse diarrhea and vomiting. Other indications are quoted from the literature. Frequent hemoglobin estimations, red, white and differential counts, urea estimation and examination of blood smears are urged, but the authors emphasize careful clinical observation. Nearly all patients had received other forms of antisyphilitic treatment, including tryparsamide, before the malaria treatment; a rather pessimistic view is expressed as to their efficacy.

Seventy-nine patients were inoculated; in six there was failure to reproduce the disease; in eight there was spontaneous abortion of the infection or complications arose before chills had occurred in five of the number. This left

sixty-five cases on which to base results; of these thirty-one were cases of general paralysis, two of juvenile general paralysis, thirteen of taboparalysis, twelve of tabes and seven of cerebrospinal syphilis. In eight cases of general paralysis complete remissions were obtained; in eight moderate improvement; in four slight improvement, and in seven no improvement. The mental features are mostly likely to improve, the neurologic elements being resistant. There were four deaths. Changes of the spinal fluid, as shown by the colloidal gold curve, were marked in the benefited patients. Three patients with taboparalysis were greatly improved; three were moderately improved; three slightly so, and four were unchanged. One patient died. One patient with general juvenile paralysis was slightly improved, the other unchanged. Three patients with tabes obtained complete subjective relief; four were improved to a considerable extent; two were slightly improved, and two did not show any change. In five cases of cerebrospinal syphilis the fluid changed to or approached the normal, although intensive antisyphilic treatment had failed to accomplish this result. The patients in two asymptomatic cases were not examined after the treatment.

Details of deaths and serologic data are given. The authors realize that only from one to nine months had elapsed before the report, but believe there is an early improvement and that further use of the method is indicated in selected cases of syphilis of the central nervous system which no longer show improvement under the usual methods of therapy.

CHAMBERS, Syracuse, N. Y.

BRACHIAL PLEXUS OBSTETRICAL PARALYSES AND REPORT OF A CASE. VINCENT GILIBERTI, J. Nerv. & Ment. Dis. **66**:449 (Nov.) 1927.

Giliberti states that this type of injury, the second largest of the birth palsy groups, may occur in normal confinement in rare instances when a combination of contracted pelvis, a large binacromial diameter and prolonged labor exists, but is produced most frequently in those cases requiring artificial aid, and in head presentations more often than those of breech, shoulder or foot. Abduction, elevation and backward movement of the arm while the head is bent toward the opposite side is the manipulation that is most apt to produce injury to the brachial plexus. Pressure on the plexus by traction with a finger or hook in the axilla and traction exerted on the shoulder to accelerate the delivery of a head are also sources of danger. The nerve fibers and bundles are usually torn and ripped apart; the break may occur at any point from the spinal origin of the roots to the lower part of the plexus. When rupture of the nerves has occurred the ends retract and scar formation prevents regeneration. The distal portion degenerates in its entirety, the central portion only at the severed end with neuroma formation. The changes of the spinal cord are slight, but are most marked in cases of avulsion of the root.

The upper arm type of paralysis, due to injury of the fifth and sixth cervical roots, is most common and involves the deltoid, brachialis anticus, biceps and supinator longus. The lower arm type of paralysis, lesions involving the seventh and eighth cervical and the first thoracic roots, affect the triceps, the flexors of the wrist, pronators, flexors and extensors of the fingers and small muscles of the hand. When the lesion is close to the vertebral foramans of the eighth cervical and first thoracic a Horner's syndrome is also produced. The combined type of paralysis is due to a lesion of the entire plexus or most of it and is probably most often caused by dislocation of the shoulder with pressure on the plexus.

Electrical reactions assist in determination of prognosis; if electrical reactions are normal or but slightly altered ten or fifteen days after onset, complete recovery should occur within six weeks; if partial reaction of degeneration is found, recovery should be complete in three or four months; a complete reaction of degeneration means a guarded prognosis as complete

recovery will not occur in all cases. The author thinks that the prevention of obstetric paralyses is, in a large measure, feasible.

Treatment in early stages is demobilization of the injured arm by means of a splint from hip to axilla which holds the arm elevated in abduction at an angle of 90 degrees. This causes approximation of the torn ends, and should be continued until function is restored. Massage, heat, hydrotherapy and exercises should be used during this time but in such a way as not to interfere with the postural treatment. If signs of regeneration have appeared within one year the mechanical and physical treatment should be continued; if not, neurosurgery should be resorted to. The matter of habit paralysis is considered. Contractures should be corrected by gradual and graded stretching, using an adjustable splint for this purpose and avoiding forceful rupture of adhesions. The case presented is interesting, but mention is not made of the treatment that was instituted in this instance.

KUBITSCHEK, Philadelphia.

CISTERNAL INJECTION OF GENERAL PARALYTIC AND TABETIC SPINAL FLUIDS INTO RABBITS. H. PETTE, Ztschr. f. d. ges. Neurol. u. Psychiat. **108**:532 (May) 1927.

Hoff and Pollock succeeded in producing changes characteristic of tabes and general paralysis in rabbits by the subdural injection of paralytic and tabetic spinal fluids. "The paralytic animals developed the typical fluid reaction of paralysis, while tabetic animals showed only a syphilitic curve in the fluid with eventual increase in globulin and cells. Animals with secondary syphilis showed a completely negative spinal fluid. Histologically, the animals with paralysis showed a meningo-encephalitis, or in other words paralysis. Animals with tabes showed slight changes. Animals with secondary syphilis did not show any histologic symptoms." Hoff and Pollock made extensive studies and concluded that "it was possible in rabbits, by subdural injection of paralytic fluid to produce a pathologic change in the spinal fluid characteristic of paralysis, and at the same time as this fluid change there occurred a severe disease of the central nervous system." They concluded that the more severe the fluid changes, the more severe are the reactions in the nervous system. They injected five animals with tabetic fluid and obtained a positive result in only one. The results were sufficiently important and conclusive to demand confirmation and this Pette set out to do.

Pette injected six rabbits cisternally with fluids from three patients with paralysis. Only rabbits with normal spinal fluids were used. The fluid was examined daily after the injection had been made. One animal died shortly after injection. All other animals showed slight transitory changes in the fluid. There occurred a lymphocytosis twenty-four hours after the injection; in one case there were 124 cells. Simultaneously an increase in protein occurred. After a few days these changes disappeared and the fluid became normal. In two animals the fluid remained unchanged. In three animals the fluid which had become normal later showed changes: cell increase, and protein increase. These animals died from six to eleven months after infection. Histologically, they showed the picture of a spontaneous encephalitis, granulomas in the cortex; slight meningeal and perivascular infiltration in the vicinity of the granulomas, and slight cell collections in the meninges. In the other animals there were not any fluid nor histologic changes.

Six animals were injected with tabetic fluids by the cisternal route. Two fluids showed positive reactions with a slight lymphocytosis which later disappeared. Histologically, there was not any disease changes noted.

Pette fails to confirm the work of Hoff and Pollock which had not been substantiated by Plaut, who also repeated them. The latter injected eight rabbits, four subdurally and four cisternally, with fluid from three patients suffering from paralysis and obtained negative results. Pette considers the

cellular reaction in the days following injection as due to the irritative action of the proteins in the human spinal fluid on the meninges of the rabbits. The reactions which occurred later in the experiments were due to the spontaneous encephalitis produced in the brains of the rabbits. ALPERS, Philadelphia.

THE BIPARTITION OF THE GRANULE LAYER OF THE VISUAL CORTEX, AND ITS IMPORTANCE IN BINOCULAR VISION. R. BÁRÁNY, Riv. oto-neuro-oftal. **4**:141, 1927.

It is known that the optic nerves undergo partial crossing in the chiasma and then run to the external geniculate bodies. These bodies are laminated; removal of one eye is followed by characteristic degeneration in alternate strata differing on the two sides. For instance, according to Minkowsky, after extirpation of the left eye there is atrophy of the first, third and fifth layers of the left external geniculate and of the second, fourth and sixth layers of the right external geniculate body. One must conclude, therefore, that the crossed and uncrossed impressions are isolated from one another in the geniculate body, and that the combination can take place only in the cortex.

The visual cortex is extremely sharply defined and is different from all other areas, owing to the duplication of the internal granule layer, the striation of Gennari or Vicq d'Azyr falling between them. This striation contains not only fibers of the optic radiation, but also short association fibers, as is shown by the fact that it is not atrophied even in persons who have been blind since infancy. The fibers of the optic radiation terminate in the deeper layer of granules.

In seeking the most simple and plausible explanation of a continuous visual field, the hypothesis is advanced that the elements which are excited by the crossed fibers and those by the uncrossed fibers are situated one above the other. If they were side by side, no continuous visual field would be possible. The two portions of the lamina granularis interna might be considered to represent anatomically the two monocular visual fields. In this case the lamina granularis would be reduplicated only where crossed fibers do not exist. In lower mammals the uncrossed fibers are few in number, and it is in these animals that the reduplication of the lamina is absent, only the inferior portion of it being found on which lies the white line of Gennari. According to Cajal and Brodmann, the reduplication of the lamina granularis is not present in the cat, but this deserves confirmation.

Basing a hypothesis on the facts mentioned, the conclusion is reached that the homolateral fibers are in the superficial stratum of the lamina granularis, since in animals without binocular vision this particular layer is absent. There is an objection, however, to this hypothesis since neither layer shows any particular atrophy following extirpation of an eye, and also because the thickness of the two laminae is different, the cells being much more numerous in the deeper division. A more plausible hypothesis, therefore, would be that all the fibers conveying crossed and uncrossed impressions end in the deep layer of the lamina granularis interna and that the external layer serves to synthesize the impressions into a harmonious continuous whole.

FREEMAN, Washington, D. C.

NEUROLOGIC MANIFESTATIONS IN PERNICIOUS ANEMIA; A NEW CONCEPTION RELATIVE TO THE ETIOLOGY. A. L. SKOOG, J. A. M. A. **87**:1957 (Dec. 11) 1926.

Addison first described pernicious anemia in 1849, and Biermer presented a good description of the disease in 1872. They described particularly the blood and gastro-intestinal symptoms. The first real conception of involvement of the central nervous system was reported by Lichtheim in 1887. Wolt-

mann states that the central nervous system is involved in 86 per cent of cases, but the author believes this is a low estimate.

A brief description is given of the gastro-intestinal and blood involvement and is followed by consideration of the neural manifestations under three heads: (1) spinal cord; (2) peripheral nerves, and (3) brain. Tingling and numbness are among the first symptoms noticed. Sensibility of the bone and joint then show impairment. Incoordination, involving early and to a greater extent the lower extremities, is recorded. One of the earliest changes is a funicular degeneration of the central portion of the posterior columns. Involvement of the spinocerebellar and crossed pyramidal tracts is frequent. All the long pathways may eventually exhibit abnormality. The absence of inflammatory evidence is striking. There is some dispute as to the frequency of true neuritis in pernicious anemia, however, many cases are on record and some have been proved by necropsy. Mild, sometimes indescribable changes in the mental state are often noted. A major psychosis is infrequent. Pathologic changes are not as striking as the changes of the spinal cord.

The author presents a case with a brief history and a report of necropsy with photomicrographs. The point of unusual interest is the extensive involvement of the anterolateral columns. The case otherwise presents the usual picture and history.

The grave prognosis is mentioned and the fact that remissions are fewer and shorter once a well defined subacute degeneration of the spinal cord has been established. The etiology is discussed and the uncertainty regarding the cause of this disease is stressed. Dr. Skoog then offers a theory that pernicious anemia has its origin in some deviation from the normal in the vegetative nervous system. He places particular emphasis on the solar plexus and calls attention to the influence of emotional strain.

CHAMBERS, Syracuse, N. Y.

THE TANGENTIAL OR INTERCALARY GANGLION OF SOME REPTILES. R. SEGARRA, Trav. du labor. de recher. biol. de l'Univ. de Madrid **24**:253 (Dec.) 1926.

This article reports a study of the intercalary ganglion in embryos of several South American snakes, chiefly *Listrophis* and *Phylodrias*. Cajal's neurofibrillar methods were used for the preparation of the slides. In the embryos studied the intercalary ganglion is well developed twenty-five or twenty-six days after the egg was laid. The nucleus has rhomboidal shape and its cells are intercalated among an important bundle of axons (central prolongations of the bipolar cells of the ganglion of Scarpa). Owing to this position the cells of the intercalary ganglion are arranged in linear series, radially oriented. While in fishes and birds the ganglion under discussion is superficial, in the snake it is well submerged in the medulla oblongata.

The intercalary ganglion consists of two lobes: (a) an upper and posterior lobe, and (b) a lower and anterior lobe. Its neurons are large, round or oval, with a pale neurofibrillar network. Most of these cells are monopolar; bipolar cells are occasionally present. The axons are parallel to the vestibular neuraxons arising in the ganglion of Scarpa. The axons from neurons in both lobes of the intercalary ganglion form a single bundle that runs almost horizontally toward the midline, crosses the raphe and is lost in the subventricular white matter of the opposite side of the medulla.

The axons of the cells of the ganglion of Scarpa form synapses with the cells of the intercalary ganglion. The chief characteristic of this synapse is the unusual shape of the axonic end. It appears under slightly diverse forms, but usually adopts the shape of a cuplike enlargement closely applied to the cell body of the intercalary neurons. The axonic swelling is often in contact with one third or even one half of the cell body of the neuron, and in many

cases is as large or even larger than the neuron. The axonic swelling is prolonged into a thin process, the central portion of the axon.

The author emphasizes the fact that since there is direct contact of the axonic swelling with the neurons of the intercalary nucleus the synapse described must be regarded as further proof of the lack of continuity of the neurons.

NONIDEZ, New York.

THE PICTURES OF SYRINGOMYELIA COMPLICATED BY SUDDEN PARALYSIS, TUMOR FORMATION, HYSTERIA AND SPINAL SYPHILIS. A. H. WOODS, China M. J. **40**:420 (June) 1926.

The anatomy of the spinal cord, especially of the course of the tactile and pain fibers, and the pathology of syringomyelia are reviewed. The various sensory, motor and trophic disturbances are explained on the position and extent of the pathologic process. Four cases are presented in which departures from the classical form of syringomyelia offered problems in diagnosis. The first case showed syringomyelic dissociation of sensation from the third thoracic segment downward and in addition presented the ordinary symptoms and conditions of transverse myelitis at the eighth or ninth thoracic segments. Spinal fluid was xanthochromic and suggested block; air injection showed a partial block at the eleventh and complete block between the ninth and the seventh thoracic segments. At operation, an ependymoma, 1.5 by 5 cm. was removed. The patient made a good recovery. Symptoms in the second case began nine months before admission to the hospital with numbness in all extremities. Six months later, sudden weakness resulted in a fall, after which the patient was paralyzed in all extremities. A syringomyelic sensory fault was found from the fifth cervical segment downward. In addition, there were symptoms of the posterior column and the limbs were in spastic paralysis. The spinal fluid was slightly xanthochromic. Air and injections of iodized oil gave suspicion of a partial block in the upper cervical segments and tumor was considered, but hematomyelia was considered the most probable explanation for the sudden tetraplegia. Postmortem examination showed syringomyelia with considerable softening and diseased blood vessels but a large hemorrhage was not found. The third case is one in which hysterical symptoms had apparently been engrafted on the disturbance produced by the organic lesion. Case 4 was that of a patient whose history gave evidence of sensory defects for nineteen years and joint trouble for two years which had developed shortly after infection with syphilis. Sensory defect, of pain and temperature, was limited to the right side; impairment in the trigeminal area, deepening into anesthesia from the sixth cervical to the first lumbar segments. The joint disease involved the right wrist, right elbow and the lower thoracic and upper lumbar vertebrae. The blood Wassermann reaction was strongly positive. It is assumed that a syringomyelic condition had existed for nineteen years and that the joint disease was of syphilitic etiology.

KUBITSCHEK, Philadelphia.

FURTHER STUDIES ON THE MORPHOGENETIC RÔLE OF THE SOMITES IN THE DEVELOPMENT OF THE NERVOUS SYSTEM OF AMPHIBIANS: THE DIFFERENTIATION AND ARRANGEMENT OF THE SPINAL GANGLIA IN PLEURODELES WALTLI. FRITZ E. LEHMANN, J. Exper. Zool. **49**:93 (Oct. 5) 1927.

Complete removal of the somites of several segments on one side in *Pleurodeles waltli* just after the closing of the medullary folds was followed by the absence of the corresponding spinal ganglia. Little, if any, differentiation of ganglion cells took place in spite of the presence of a normal spinal cord when myotomes and skeleton were lacking completely or a grafted spinal cord was adjacent to other parts of the myotome than its median surface. Differentiated ganglion cells were found, even in the case of a defective cord,

in which the median surface of the somite was left or in which the cartilaginous skeleton was present. In regions with partial destruction of the somite boundary the ganglia are loose and elongated toward the defective boundary. A shifting of somites in regard to the longitudinal axis causes a shifting of the spinal ganglia by exactly the same amount. The somite seems to have the following properties: its median surface stimulates and its lateral surface inhibits differentiation of ganglion cells, while its boundaries determine the segmental arrangement of the spinal ganglia.

Two factors, partially overlapping in their effects, determine histologic differentiation of the medullary material in the gastrula. One of them is localized in the medullary substance, the other in the roof of the archenteron. The arrangement of the differentiating medullary substance is controlled by the underlying mesoderm. The form of the closed medullary tube is developed according to the behavior of the surrounding somites. At these early stages the quantity of the formed medullary substance is determined by the mesoderm. After closing of the folds, the spinal cord seems to become independent of the surrounding mesoderm as far as the rate of proliferation is concerned. Various factors within the spinal cord itself affect its further growth. The source of the ganglion-crest cells also seems to be determined at this time. Their differentiation and arrangement, however, are determined by the mesoderm and their rate of proliferation by the sensory area. The segmental arrangement of both motor roots and spinal ganglia is under the influence of the segmentation of the mesoderm.

WYMAN, Boston.

BILATERAL LUMBAR SYMPATHETIC GANGLIONECTOMY AND RAMISECTOMY FOR POLYARTHRITIS OF THE LOWER EXTREMITIES. LEONARD J. ROUNTREE and ALFRED W. ADSON, J. A. M. A. **88**:10 (March 5) 1927.

The authors attempted a new form of treatment in arthritis deformans. They emphasize the trophic changes present, such as cold and clammy hands and feet, pallor and cyanosis of the extremities, and puffiness of the tissue. Other forms of arthritis may manifest partial ischemic rather than strictly trophic changes.

Analysis of older methods of treatment shows that all methods aim at increasing the circulation of the joint or extremity. Such an increase, marked and lasting, follows lumbar sympathetic ganglionectomy and ramisectomy in other diseases. This procedure was adopted in a case of polyarthritis, which is reported. The patient was a single woman, aged 34, whose history was unimportant except for generalized chronic arthritis from which she had suffered for six years; finally, she had become incapacitated for work. Physical examination revealed only the usual symptoms in generalized chronic arthritis of moderate or severe degree.

The surgical procedure adopted consisted in the removal of the second, third and fourth lumbar sympathetic ganglia on both sides with the sympathetic trunk. All the rami to the hypogastric plexus were divided, as well as those passing to the second, third and fourth lumbar nerves. The rami to the sacral nerves were not divided. The patient's feet have become dry, the skin has desquamated and the trophic changes have largely disappeared since the operation. The pain has disappeared and has been replaced by a pleasant sensation of warmth. In contrast to this the hands, wrists and shoulders are continually painful, cold and clammy and measure 9 calories lower than the feet. A table of studies of the temperature of the skin and elimination of heat of the left foot is presented.

Only immediate results can be given now but they are most encouraging. The patient has requested cervical sympathetic ganglionectomy for relief of the arms and shoulders, but this has not been done because results are not so favorable as with the lumbar operation for relief of the lower extremities.

The authors believe it wiser to defer further operative treatment pending observation of the results of the operation already performed.

CHAMBERS, Syracuse, N. Y.

A TECHNIC FOR THE STAINING OF THE NERVOUS SYSTEM WHILE ENCLOSED IN ITS BONY CAPSULES. F. DE CASTRO, Trav. du labor. de recher. biol. de l'univ. de Madrid **23**:427 (May) 1925.

Pieces of vertebral column, skull, joints and other parts of mice and other small mammals, young or almost adult, are preserved in any of the following mixtures:

Chloral Hydrate Mixture

Chloral hydrate	2–5 Gm.
Distilled water	50 cc.
Pure alcohol	50 cc.
Nitric acid	3–4 cc.

Uretane Mixture

Ethylic uretane	1–2 Gm.
Distilled water	40 cc.
Pure alcohol	60 cc.
Nitric acid	3–4 cc.

Somnifene Mixture

Somnifene (Hoffmann-LaRoche)	2–4 cc.
Pure alcohol	60 cc.
Distilled water	40 cc.
Nitric acid	3–4 cc.

The length of preservation varies from one to three days, according to the size of the pieces. Large blocks should be cut into slices from 4 to 5 mm. thick after they have been in the preserving fluid from twelve to eighteen hours. Wash in distilled water (from twenty-four to thirty-six hours) to remove nitric acid. Transfer to alcohol-ammonia (from twenty to twenty-four hours), then proceed as for Cajal's method (1.5 or 2 per cent silver nitrate solution for from five to seven days in the oven at from 37 to 40 C.; distilled water; reduction in formaldehyde-pyrogallic acid mixture, etc.). Embed in celloidin or paraffin. The sections may be toned with gold chloride if desired.

The results of application of this technic to the medulla, spinal cord, sympathetic ganglia, and labyrinth of the mouse are illustrated with excellent photomicrographs.

NONIDEZ, New York.

THE SYMPTOMATOLOGY AND PATHOGENESIS OF PYKNOLEPSY. JAKOB RATNER, Monatschr. f. Psychiat. u. Neurol. **64**:283 (Aug.) 1927.

The author reports three cases, one of pyknolepsy, one of narcolepsy, and one of tonic (subcortical) convulsions with hypophysial obesity and coloboma of the iris and choroid. The case reports are unusually interesting.

He believes that pyknolepsy and narcolepsy, though not identical, are closely related, and attempts to extract the common factor of the two diseases. This factor, he believes, is a constitutional weakness of the diencephalon. To these two he adds a series of other diseases, all of which, he believes, have diencephalic insufficiency as a common basis. These diseases he groups under the name "Diencephaloses." The central syndrome in the series is the Bardet-Biedl syndrome (hypophysial obesity with polydactylia and retinitis pigmentosa sine pigmento). The group also includes the "arthritism" of the French (obesity, diabetes, gout), paralysis agitans (?), certain forms of manic-depressive

insanity, Bratz' "affectepilepsy," Oppenheim's psychasthenic convulsions, catatonic attacks (?), and hysteria (?). (The question marks are the author's).

In comparing pyknolepsy and epilepsy, he points out that there are no psychic anomalies in true pyknolepsy — no deterioration and no character changes; and he explains this on the basis of its subcortical origin. The inhibition of cortical function in pyknolepsy does not originate in the cortex, but is imposed on the cortex by impulses from the diencephalon. In true epilepsy, on the other hand, the stimulus starts in the cortex and affects the lower centers secondarily. The essential process in pyknolepsy is corticopetal; in epilepsy it is corticocentric and corticofugal.

He describes two things more or less typical of the diencephaloses: (1) certain anatomic changes in the sella demonstrable by roentgen ray ("closed sella," dorsum elongatum and small shallow sella), and (2) a diminished pyrogenetic reaction as evidenced by hypothermia and absence of fever after the intramuscular injection of 10 cc. of boiled milk.

SELLING, Portland, Ore.

THE EFFECTS OF FUNCTIONAL UNION OF THE CENTRAL END OF THE PHRENIC NERVE WITH THE PERIPHERAL END OF THE MOTOR NERVE TO THE STERNOHYOID MUSCLE. NARCISO CORDERO and A. J. CARLSON, Am. J. Physiol. **82**:580 (Nov.) 1927.

The authors' study was carried out on a series of five dogs in which the central end of the upper phrenic root was sutured to the distal end of that branch of the spinal accessory representing the motor supply of the sternohyoid muscle on the same side. This procedure was completely successful in four of the five dogs as determined by surgical exploration from 104 to 182 days after operation. In all four of these cases in which successful functional union had occurred homolateral contractions of the muscle were consistently observed in association with each inspiration and varying in degree with respirational depth. These contractions were not of sufficient magnitude, however, to be visible through the skin but were noted only after the muscle had been exposed and separated from its heterolateral mate. Section of the nerve was followed by cessation of contraction, with peripheral stimulation positive for contraction and central stimulation negative. Also, for the observation periods utilized, there was not any evidence of discharge modification or division by the respiratory center in obviation of the noted and essentially useless sternohyoid contractions. From this work it is apparent that cross anastomosis between nerves of different function may result in functional restoration, although it does not appear that there also occurs a restoration of coordination normality as shown by the absence of superfluous movements. At this point the authors raise the interesting question as to whether or not the elimination of such extra movements may not be brought about through a process of reeducation. The principle involved in this experiment, it might be added, is of no little clinical significance, for example in the spinal accessory — facial anastomosis in the treatment of facial palsy.

RAPHAEL, Detroit.

A GROUP OF CASES WITH SYMPTOMS OF DISSEMINATED ENCEPHALOMYELITIS. EMIL REDLICH, Monatschr. f. Psychiat. u. Neurol. **64**:152 (July) 1927.

Redlich reports a group of cases, most of them observed within a short space of time, which he classes as disseminated encephalomyelitis. Some of the cases presented pure spinal lesions (transverse myelitis or Brown-Séquard syndrome), others a combination of spinal and cerebral symptoms. The onset was acute or subacute, the course brief, and the outcome generally favorable. Many of the patients recovered completely, others had slight permanent residual defects; in a few, the residual defects were marked. The point of the article is in the grouping of the cases. The possibility of acute multiple sclerosis is

discussed in detail, but discarded. Epidemic encephalitis is also considered. In one or two patients the onset was suggestive, but, on the whole, the cases were not typical and the author concludes that they are outside the lethargic group. He points out the frequency of recent reports of acute encephalomyelitis accompanying varicella, variola, vaccination, measles and other diseases. Because of this, he considers the possibility of an activation of some latent neurotropic virus.

From this article, as from the clinical experience of recent years, one must conclude: that the group of acute and subacute, patchy, scattered lesions of the central nervous system, including the various types of disseminated encephalomyelitis, epidemic encephalitis, multiple sclerosis and perhaps Schilder's disease, form a field in which the boundaries are often difficult to define — not only on clinical, but also on pathologic grounds; that there are cases which cannot be satisfactorily cataloged, and that further etiologic and pathologic studies must be awaited for an accurate differentiation.

SELLING, Portland, Ore.

ENCEPHALITIS AFTER VACCINATION. D. WIERSMA, Acta Psychiat. et Neurol. **2**:167, 1927.

The author, in reporting eleven cases of encephalitis after vaccination calls attention to the fact that sixty or eighty cases have occurred. There was considerable difference in opinion, however, among the investigators who described these cases as to the nature of the disease. Some believed it to be ordinary epidemic encephalitis; others reported varying symptomatology. The mortality was around 70 per cent.

The cases described by Wiersma developed from nine to nineteen days after vaccination. The first symptoms were weariness and drowsiness, but soon high fever, convulsions, paralyses and Kernig's sign occurred. Trismus was noted in some cases; the Babinski sign was almost always present, and ocular paralyses were not observed. The patients lay in coma rather than in sleep. Sequelae were not observed in the patients that recovered.

There is also some disagreement as to the anatomic aspect, since Lucksch described conditions typical of epidemic encephalitis, whereas Bastiaanse noted characteristic differences. "The alterations in encephalitis after vaccination are localized mainly to the white substance," and the basal ganglia and locus niger are spared. The pathogenesis of the disease is not clear. It was thought by some that the vaccine virus was contaminated; by others, that the patients harbored the virus of encephalitis, which became active following the lowered immunity resulting from the induced infection. It may be that some new virus activated by the vaccine virus is yet to be discovered.

FREEMAN, Washington, D. C.

EXPERIMENTAL STUDIES ON MAUTHNER'S CELL IN AMBLYSTOMA. S. R. DETWILER, J. Exper. Zool. **48**:15 (July 5) 1927.

The two giant Mauthner neurons which occur in the central nervous system of teleost fishes and of amphibians exhibit certain characteristic anatomic features. Chief among these are the gigantic size of the cell, the high degree of differentiation, the characteristic position in the medulla, the very extensive dendritic connections, the internal structure, and the nature of its synapses. Mauthner's cell can be excised in the embryos of *Amblystoma* without creating any other noticeable defects in the central nervous system. Larvae lacking one or both Mauthner axons may exhibit essentially normal posture at rest and during activity. Marked equilibratory disturbances in larvae lacking Mauthner's cell are in all cases associated with defects in the ear or its nervous mechanism resulting from the operation. Larvae lacking one or both Mauthner's neurons exhibit short, jerky, poorly coordinated swim-

ming reflexes which they are incapable of sustaining. They exhaust rapidly. A tendency toward rapid exhaustion and general inactivity is seen also in larvae lacking one ear. This is no doubt associated with the lowered tone of the muscles. The results of the experiments indicate that: unless some other tract compensates for its absence, Mauthner's neuron in *Amblystoma* is not primarily concerned with maintaining equilibration. That it plays an important part in transmitting impulses to the primary motor centers which are responsible for coordinating and sustaining the swimming reflexes is apparent from the type of activity in animals lacking this neuron.

WYMAN, Boston.

THE PROGRESS OF TETANUS. ASTLEY P. C. ASHHURST, J. A. M. A. **87**:2089 (Dec. 18) 1926.

Ashhurst believes that physicians should be warned not to be misled by Wainwright's recent paper with its conclusion that intraspinal injections of antitoxin are harmful in the treatment of tetanus, increase mortality and should be abolished. Ashhurst believes that antitoxin injected intraspinally has a specific action on the toxin in the nerve roots and cord. He discusses the subject under the headings of: (1) intraspinal intravital stains, methylene blue injected subdurally in the lumbar region of rabbits having been found in the optic tracts (anterior end) and, in patches, in the cerebrum; (2) difficulty of medicating the cord by intravenous methods, and (3) the clinical results of intraspinal injections of antitoxin. Ashhurst has had twenty-three cases with eight deaths or 34.2 per cent mortality; if only patients seen early and treated efficiently are counted, there are eighteen cases with three deaths, or 16.6 per cent mortality. Early diagnosis is most important. Ashhurst: (1) abolishes the source of toxin (infected wound); (2) gives from 15,000 to 20,000 units of antitoxin intravenously; (3) injects from 6,000 to 10,000 units intraspinally immediately; (4) administers spinal depressants until physiologic effects are produced, and (5) gives careful feeding and nursing.

CHAMBERS, Syracuse, N. Y.

STUDIES IN ANIMAL AGGREGATIONS: SOME PHYSIOLOGICAL EFFECTS OF AGGREGATION ON THE BRITTLE STARFISH, OPHIODERMA BREVISPINA. W. C. ALLEE, J. Exper. Zool. **48**:475 (Aug. 5) 1927.

Animals frequently collect in nature and in the laboratory so that they occupy only a fraction of the available space. Such collections may be called animal aggregations. *Ophioderma brevispina* forms aggregations in the laboratory in the most shaded region or around some quiet individual. The aggregations form in the main by individual (primary) responses to environmental conditions rather than by social (secondary) impulses. The initial effect of such aggregation is a lessening of the rate of oxygen consumption, probably caused by decreased muscular tonus. In the absence of food and with the proper volume of water, such aggregations soon come to consume oxygen at a higher rate than do a similar number of individuals isolated in the same volume of water per individual, and in the end the members of a group have a greater average survival time than their isolated fellows. Similar effects are observed when *Ophioderma* are isolated in containers supplied with glass rods among which the isolated individuals can come to rest. It appears that the greater survival value of the aggregation is due, at least in part, to the substitution of other individuals for elements of the normal physical environment. The possibility of autoprotective secretions is also considered. The survival value of the group may be so neutralized by adverse conditions that the group may have the deleterious effects usually attributed to crowding.

WYMAN, Boston.

A Case of Right Sided Hemianopia and Alexia with Monocular Diplopia of Central Origin. F. Quensel, Monatschr. f. Psychiat. u. Neurol. **65**:173

The author presents a carefully studied case of gunshot wound of the skull involving both occipital lobes. The patient showed: right homonymous hemianopia, with a scotoma in the relatively intact left field; alexia, and monocular diplopia. The monocular diplopia existed in each eye, was symmetrical and present only in a limited part of the visual field. It was constant. The object fixed was seen clearly and sharply in its proper place; the second image was somewhat different and blurred and from 25 to 30 degrees away. Monocular diplopia is relatively rare. It occurs in hysteria and after operations for strabismus. As a tentative explanation of its occurrence in this case, the author offers the fact that the functional capacity of the two hemispheres must have been different. The images from the two sides consequently, were not fused, but came to consciousness independently. The greater damage was done to the left hemisphere. From the course of the bullet, the left optic radiation and calcarine region and probably the adjacent portion of the parietal lobe must have been badly injured. The right occipital lobe was damaged, but much less extensively. From the situation of the scotoma, probably the upper calcarine lip was involved. The right hemisphere gave a relatively normal picture; the blurred, displaced image probably came from the left hemisphere.

Selling, Portland, Ore.

"Narcosan" and Drug Addiction. Current Comment, J. A. M. A. **87**:2097 (Dec. 18) 1926.

On Dec. 15, 1926, newspapers carried a story concerning "narcosan," which was discovered by "Dr." A. S. Horovitz. Since 1913, this man has been continuously identified with attempts to promulgate cures for all sorts of disorders by the administration of mixtures of lipoids and nonspecific protein vegetable substances; one of these, introduced in 1920 as "lipoidal substances," has recently been called "narcosan." The present effort to promote "narcosan" as a cure for narcotic addiction is supported by a clinical investigation by Drs. Alexander Lambert and Frederick Tilney. Their report appears in the M. J. & Record **124**:764, 1926. This paper had been rejected by the *Journal of the American Medical Association* because the Council on Pharmacy and Chemistry had previously rejected the product in 1921, because it has not been resubmitted to the council, and because of the lack of evidence of ordinary controls. Simultaneously with the publication of the article, a highly exaggerated statement was issued by the North American Newspaper Alliance.

Time may reveal sufficient basis for the acceptance of the Horovitz discovery; the work of Lambert and Tilney may have been strictly accurate and scientific, but emphasis is laid on the safe, scientific method of introducing a new proprietary remedy through its presentation and acceptance by the Council.

Chambers, Syracuse, N. Y.

Central Factors in Hunger. Frederick Hoelzel, Am. J. Physiol. **82**:665 (Nov.) 1927.

A full discussion relating to the theory of hunger, based on certain personal experiences of the author, and a careful survey of the literature is presented in this paper. The conclusion is reached that, fundamentally, hunger is centrally rather than peripherally or gastrically determined. This belief is based first on the observation that when desire to eat is suppressed by excessive food ingestion, the following gastric (hunger) contractions are noted as local (epigastric) feelings unattended by "hungriness," also the fact that hunger is noted to occur after fasting independently of gastric movement. The common reference of hunger to the stomach is dependent more on the epigastric seat of the so-termed incidental hunger factors, as gastroduodenal acidity and gastric emptiness in relation to periodic contraction of the stomach, rather than

on such locale for the specific phenomenon or urge of hunger as such. This conception of an essentially central source or origin for hunger is interesting and renders more easily understood such observations as the lack of hunger in certain animals during sexual heat (in seals and salmon), the persistence of eating when the stomach has been removed, and the commonly observed hunger-inhibiting effect of fever, affective activity and psychic preoccupation.

RAPHAEL, Detroit.

TOXIC REACTION FROM PHENOBARBITAL (LUMINAL): REPORT OF SIX CASES. ARNOLD S. JACKSON, J. A. M. A. **88**:642 (Feb. 26) 1927.

In spite of the widespread use of phenobarbital, the literature contains only meager reports of cases of poisoning therefrom. The author was able to find only seven cases with one death. An erythematous rash and fever were the most common results. Diarrhea, vertigo, diplopia, paraphasia, dilated pupils and disturbances in speech were also observed. Six cases were observed among the 500 patients who received the drug during the preceding year. Three photographs illustrating the rash are presented. Fatalities did not occur in the group.

Jackson's conclusions are that: phenobarbital is valuable in the treatment of various nervous and mental disorders and in certain conditions, such as insomnia, occurring in association with organic diseases like toxic goiter; (2) the dangers from phenobarbital, although the drug is widely used, have scarcely been appreciated; (3) the characteristic signs and symptoms are an erythematous, maculopapular, generalized skin eruption accompanied by intense itching, fever, weakness, vertigo and malaise. Treatment is prophylactic warning, prompt withdrawal of the drug, the use of local sedatives and eliminative procedures. One and one-half grains (0.097 Gm.) before supper is a satisfactory and apparently safe dose for insomnia, and ½ grain (0.032 Gm.) three times a day for nervous states.

CHAMBERS, Syracuse, N. Y.

EXOPHTHALMIC GOITER AND THE INVOLUNTARY NERVOUS SYSTEM: XIII. THE COURSE OF THE SUBJECTIVE AND OBJECTIVE MANIFESTATIONS OF EXOPHTHALMIC GOITER IN FIFTY UNSELECTED PATIENTS. LEO KESSEL and HAROLD T. HYMAN, Arch. Int. Med. **40**:314 (Sept.) 1927.

A series of fifty patients with exophthalmic goiter was divided into subgroups: (a) those seeking specific therapy, dying, or lost to view; (b) those in whom the disease took a "spontaneous course." By this term is meant the absence of specific thyroid therapy, operation and other procedures. Thirty-one cases fell into this second group, and the patients have been followed, on an average, for almost five years. While symptoms such as rapid pulse, tremor, and enlargement of the thyroid have not disappeared, there was observed an almost steady diminution of the basal metabolic rate. Best of all, a return to economic and social duties was encompassed in the vast majority in this group. The conclusion is reached that "with regard to the spontaneous course we may conclude that in considerably more than half of the patients economic and social restitution may be expected after the fourth month which continues up to at least on an average of fifty-seven months, and this restitution is made in the presence of residual symptoms and despite the stress and strain of the needy in a metropolitan district."

ANDERSON, Philadelphia.

POLYNEURITIS IN PERNICIOUS ANEMIA. LUDO VAN BOGAERT, Ann. de méd. **22**:321 (Oct.) 1927.

Involvement of the peripheral nerves in pernicious anemia seems to be more frequent than would be demonstrated by the few cases published. According to Pierre Mathieu, clinical diagnosis is extremely difficult on account of the simultaneous affection of the spinal cord. Early involvement of the muscle

groups at the antero-external side of the legs may help to establish the differential diagnosis. One out of six cases of pernicious anemia with disease of the nervous system presented a picture of polyneuritis, and this was verified histologically. A woman, aged 22, developed a rapidly progressive pernicious anemia; within fifteen months she had polyneuritis of all extremities. The sensory disturbances affected deep sensibility, and sensation for temperature, pain and finally touch. Diffuse muscular atrophy took place in the lower extremities. Later, mental confusion developed with paranoic features. Histologic examination showed the spinal cord intact. In the right peroneal nerve, atrophy and degeneration of nerve fibers were found together with cellular infiltration and beginning of thickening of the peri-arterial connective tissue. Similar lesions were found in the crural, sciatic, posterior tibial and left radial nerves. The peroneal muscles showed interstitial myositis.

WEIL, New York.

A COMPARATIVE STUDY OF THE EXTENT OF THE KNEE-JERK AND THE ACHILLES-JERK. W. W. TUTTLE and L. E. TRAVIS, Am. J. Physiol. **82**:147 (Sept.) 1927.

This work was carried out on a series of 122 persons (60 men and 62 women) utilizing a special apparatus devised to deliver automatically stimuli of uniform intensity and rate. The authors found the strength correlation between these two reflexes to be $+0.37$, ± 0.12 for forty-five men, and $+0.54$, ± 0.09 for fifty-four women, and $+0.42$, ± 0.06 for the joint group, indicative of little relation between the two response strengths for the same subject. Further, it was determined that the patellar response in men was only 0.5 of that of the women, and 0.7 for the Achilles jerks. Likewise, the extent of the knee jerk was noted as much more variable among women than men, but about the same for the two sexes as respects the Achilles response. Also of interest is the observation that, with the excitation unit employed, there was not any Achilles jerk found in fifteen men and in only five women, and there was not any knee jerk in any women and in only six men. No correlation, it might be mentioned, was determined between either reflex response and height, weight or age.

RAPHAEL, Detroit.

VERTIGO. SURGICAL TREATMENT BY OPENING THE SACCUS ENDOLYMPHATICUS. GEORGES PORTMAN, Arch. Otolaryng. **6**:309 (Oct.) 1927.

Portman calls attention to the fact that pressure on the saccus endolymphaticus may cause vertigo, deafness and tinnitus. This may be from without, as from an adjacent organ from the lateral sinus or from the cerebellar meninges — or from within, as in serous labyrinthitis, leading to overproduction of the endolymph and a development of "auricular glaucoma." Relief of this pressure by puncture of the saccus may prevent the delicate end organs from receiving serious damage, and may relieve the vertigo for an indefinite period. The operative technic is described.

Fifteen years ago Bárány reported relief of vertigo caused by "adhesions." His operation had the same result of lessening intralabyrinthine tension by tearing the ductus endolymphaticus. Portman has performed the operation only when all other sources of vertigo were eliminated. Although it would appear that the relief would be temporary, Portman reports a patient cured for two years, and calls attention to similar results after puncture for hypertension in the eye.

HUNTER, Philadelphia.

EXPERIMENTAL STUDIES ON THE SUGAR CONTENT OF THE CEREBROSPINAL FLUID. C. J. MUNCH-PETERSEN and KNUD WINTHER, Acta psychiat. et neurol. **1**:209, 1926.

The authors studied the relation between the sugar content in the blood and the cerebrospinal fluid in forty-two persons. They found that a simple alimentary

hyperglycemia, even if prolonged, did not produce an increase of the sugar content of the cerebrospinal fluid. The increase of sugar in the blood following the injection of epinephrine was accompanied, however, by a similar increase in that of the cerebrospinal fluid. Similarly, if the sugar content of the blood was increased by the simultaneous injection of epinephrine and glucose, that of the cerebrospinal fluid was increased even if the amount of epinephrine injected was too small by itself to cause any change. The sugar content of the cerebrospinal fluid may be increased without the appearance of sugar in the urine. From their studies, the authors conclude that the cells that secrete the cerebrospinal fluid possess an influence determining the extent of the increase of sugar in the fluid. Epinephrine appears to irritate certain centers in the subthalamic region or brain stem which control the secretion of sugar into the cerebrospinal fluid and thus provokes an increase in the sugar content.

PEARSON, Philadelphia.

STUDIES ON THE VISCERAL NERVOUS SYSTEM: REFLEXES FROM THE GASTRO-INTESTINAL TRACT TO THE EYE. J. F. PEARCY and T. D. ALLEN, Am. J. Physiol. **82**:56 (Sept.) 1927.

Stimulated by the frequent association of gastro-intestinal "upsets" and visual disorder, the authors conducted a careful series of observations relative to ophthalmologic changes in response to stimulation of the upper and lower portions of the intestinal tract in man as induced by air inflation. They find that such distention of the stomach or intestine is definitely followed by reflex disturbance of the vision, specifically a pronounced blurring, more particularly for objects which are near. This blurring the authors have demonstrated largely depends not on ametropia, heterophoria or alteration of intra-ocular pressure, but rather on a decidedly marked ciliary hypotonia, retinal congestion and edema. To some extent, also, this noted blurring has a subjective basis—"wandering of attention." This factor is simply one aspect of the "general nervous reaction" consequent to alimentary distention identical with the general picture noted subsequent to the ingestion of a heavy meal and including also drowsiness, a feeling of relaxation, perspiration and diminution of the patellar response.

RAPHAEL, Detroit.

THE TREATMENT OF NEUROSYPHILIS BY MALARIA. EDITORIAL, J. A. M. A. **87**:2164 (Dec. 25) 1926.

There are encouraging indications of the value of the von Jauregg method in the treatment of neurosyphilis and particularly of parenchymatous neurosyphilis. Its limitations must be realized at the outset. We must have the best conditions for observation and management. The unattainable must not be expected. The success of the treatment is to be judged by the amelioration or disappearance of the symptomatic complaint and not by the objective signs alone. There are other recently introduced methods such as the use of the organism of rat-bite fever and the injection of proteins. The question at issue involves the possible necessity of having living organisms for successful therapy. Competent observers tell us that in localities where malaria is widespread syphilis of the central nervous system seems to be rare.

CHAMBERS, Syracuse, N. Y.

CASE REPORT OF WORD BLINDNESS AND NOTE BLINDNESS. OTTO POTZL, Monatschr. f. Psychiat. u. Neurol. **64**:1 (Sept.) 1927.

The author reports a patient with the following picture: word blindness, number blindness and note blindness; no agraphia; disturbed orientation on own body (autotopagnosia) with sparing of the fingers; cortical epileptic attacks, with seizures beginning in the right thumb, and associated with turning of the head, eyes and body to the right; right homonymous hemianopia.

Analysis of this and of other cases of word blindness leads the author to the view that there are two distinct lesions which may result in word blindness. One (to which this case belongs) is a parieto-occipital focus, belonging to the territory of the middle cerebral artery; the other is a focus deep in the medulla of the lingual gyrus, belonging to the distribution of the posterior cerebral artery. A discussion is given of the criteria for differential diagnosis of the two lesions.

SELLING, Portland, Ore.

TRYPARSAMIDE IN THE TREATMENT OF GENERAL PARALYSIS. SAMUEL B. HADDEN and GEORGE WILSON, J. A. M. A. **88**:473 (Feb. 12) 1927.

A brief history and description of tryparsamide are first given. Three classes of cases were treated: organic, organic reactions with psychoses of functional coloring, and transitory psychoses without signs of deterioration. Tryparsamide was given alone without mercury or bismuth. The authors consider their results encouraging and better than older methods of treatment. Clinical results surpass the serologic; the use of mercury, bismuth and other arsenicals might have produced better serologic results. The authors believe that the danger of visual damage, while it exists, has been overemphasized. Beneficial results were obtained in 40 per cent of the cases in which treatment was administered; it is the drug of choice in the treatment of general paralysis.

CHAMBERS, Syracuse, N. Y.

DISTURBANCE OF THE OPTIC NERVE CAUSED BY MALIGNANT DISEASE OF THE SPHENOID: REPORT OF TWO CASES. LEON A. WHITE, Arch. Otol. Otolaryng. 6:361 (Oct.) 1927.

White reports in detail two patients with supposed retrobulbar neuritis who were referred for examination of the sinuses as a possible focus of infection; the lesion proved to be a carcinoma. Although this is a rare occurrence it should be given consideration in cases of retrobulbar neuritis. White has found it twice in 125 cases. Meningitis was the terminal stage. Radium was advised in treatment.

HUNTER, Philadelphia.

ACUTE POLIOMYELITIS WITH AN ISOLATED PALATINE PARALYSIS. JOS. C. REGAN, J. A. M. A. **87**:1997 (Dec. 11) 1926.

A case of palatine paralysis with the usual history of infantile paralysis, to the exclusion of all other paralyses is reported. The physical examination, course and laboratory observations are presented in some detail. Only one such case is recorded in the literature (Wickman). It has been considered that the presence or absence of palatine paralysis served as a valuable point of differentiation between Heine-Medin's disease and postdiphtheric paralysis. Points of differentiation in this case are presented and the conclusion made that palatine paralysis may occur in acute poliomyelitis and be the only paralytic evidence of the disease.

CHAMBERS, Syracuse, N. Y.

LESIONS OF THE EPICONUS. B. J. ALPERS, J. Nerv. & Ment. Dis. **66**:468 (Nov.) 1927.

The clinical syndrome of the epiconus as described by Minor consists of involvement of the sacral plexus, (the nervus peroneus being most involved) absence of ankle jerks, integrity of sphincters and integrity of patellar reflexes. Three clinical cases are reported in detail, one apparently of syphilitic etiology, the nature of the pathologic change in the other two obscure. The anatomy of the epiconus region is reviewed; it included the fifth lumbar to the second sacral segments, inclusive. The literature is also fully reviewed.

KUBITSCHEK, Philadelphia.

THE TREATMENT OF NEUROSYPHILIS. J. L. MCCARTNEY, Am. J. Syph. **11**:398 (July) 1927.

This article consists of a long discussion, well outlined, of the various methods of treatment. The technic of intravenous, intraspinal, subarachnoid, cisternal and other methods is presented clearly and with a minimum of words. In this part, the material presented is mainly an assembling of more or less recognized methods; the second part will follow in the October issue.

ANDERSON, Philadelphia.

MICROSCOPIC SLIDE PRECIPITATION TEST FOR SYPHILIS. B. S. YOUNG and A. M. YOUNG, Am. J. Syph. **2**:290 (April) 1927.

This is the second communication on the microscopic slide precipitation test in which further refinement resulting in increased simplicity and specificity is reported. The technic is described in detail, and the authors conclude that this test is as specific as the Kahn and Wassermann tests; it has the advantage over those methods of being much simpler, and requires less apparatus and a smaller quantity of serum.

KUBITSCHEK, Philadelphia.

TRYPARSAMIDE IN ACUTE SYPHILITIC MENINGITIS. E. R. SMITH, Am. J. Syph. **11**:433 (July) 1927.

This article consists of brief reports on nine cases of acute syphilitic meningitis. Five were cases that occurréd during or soon after the secondary infections; four were characterized as latent, the symptoms following trauma to the head. In all cases, prompt and effective relief of discomfort followed the use of this drug, without the occurrence of any untoward complications.

ANDERSON, Philadelphia.

ENLARGEMENT OF THE CORTEX IN EXPERIMENTAL UREMIA. E. M. MACKAY and L. L. MACKAY, J. Exper. Med. **46**:429 (Sept.) 1927.

The production of an experimental uremia in the albino rat by the removal of both kidneys is followed by hypertrophy of the suprarenal cortex, which amounted to approximately 40 per cent of volume in males and 61 per cent in females.

FREMONT-SMITH, Boston.

Society Transactions

SECTION ON NEUROLOGY AND PSYCHIATRY OF THE NEW YORK ACADEMY OF MEDICINE

Regular Meeting, Oct. 11, 1927

WALTER M. KRAUS, M.D., *Chairman, in the Chair*

A CASE OF PROFOUND EMACIATION OF UNKNOWN ORIGIN. DR. JOHN MCD. MCKINNEY.

Elizabeth H., aged 29, single, entered the tuberculosis ward of the Bellevue Hospital on June 7, 1927, complaining of extreme weakness and emaciation, gas and pain in the stomach and cough with expectoration since June, 1926. She had had bronchitis frequently when a child, but no operation nor any previous serious illness. The patient left school in the eighth grade at the age of 15 and has worked ever since. Menstruation began when she was 14, and occurred every twenty-eight days, lasting from four to five days; it was copious, and was accompanied by pain and occasionally by nausea and vomiting on the first day. The periods, were regular until the onset of this illness. She has not menstruated since.

The patient was taken sick in June, 1926, with headache, fever, general malaise, redness and pain and swelling of the right knee. Following this, the small joints of the right hand became involved, and a little later pain and swelling appeared in the back of the neck. The patient was confined to bed. On Aug. 5, 1926, she was sent to the St. Francis Hospital, and she remained there until September 20. During the stay in the hospital she had a fever continuously, often as high as 104 or 105 F. The patient was delirious, had myoclonic twitching of the face, trunk and extremities and occasionally saw double. She had almost constant nausea and vomited frequently. She lost a great deal in weight and became extremely weak. Stiffness developed in the hands and legs. She also developed bed sores over the sacrum and the right hip. On September 20, the patient left the hospital against advice and was taken home. The roentgenograms of the chest and examination of the sputum were negative for tuberculosis. The spinal fluid was normal. The Wassermann reaction and blood cultures were negative. A tentative diagnosis of acute pulmonary tuberculosis was made. She remained at home from September, 1926, until June, 1927, when she was admitted to the Bellevue Hospital. The temperature continued elevated for the first month of this period and then gradually subsided. She continued to have frequent attacks of vomiting, frontal headache, dizziness, diplopia, myoclonia and increased salivation. During this period she developed great difficulty in sleeping at night. However, she did show a very gradual amelioration of the symptoms. She entered the tuberculosis ward at the Bellevue Hospital June 7, 1927, and as no laboratory or clinical evidences of tuberculosis or other visceral pathology was found to account for the symptoms, she was transfered to the neurologic service on June 20.

Examination.—Physical examination at the time of admission revealed a much emaciated woman. She was too weak to sit up, stand, walk or even feed herself. The skin was dry and there was scaliness of the scalp and lips. The hair and nails were dry and brittle. The teeth were long and angular, with spaces between the upper central and lateral incisors. The lower arch was narrow, the teeth were crowded and there was some malocclusion. The hair distribution was of the feminine type, and the skeletal conformation belonged to the asthenic group. The skin was inelastic, there was no panniculus and the muscles were much shrunken, giving the impression of nothing but

skin and bones. There were scars of healed bed sores over the sacrum and right hip. The tonsils were buried, but enlarged. The pulse rate was 100 and respiration rate 24. The weight on admission was not determined because she was too weak to be moved. The first recorded weight was on July 13, when she weighed 65 pounds (29.5 Kg.). Her normal weight before the illness was 122 pounds (52.3 Kg.).

Course and Treatment.—On admission to the neurologic ward the patient was given a high caloric diet, her bed was placed on the porch and she was put in the sun whenever possible. Medication consisted of malt and codliver oil, 2 drams three times a day. She had a slight elevation of temperature during the first week and was irrational at times. She took nourishment fairly well. Nausea gradually subsided and the vomiting practically stopped after the first week, and she has been gradually gaining in weight and strength since. The dryness of the skin and the scaliness of the scalp and lips gradually cleared up, and the patient became able to feed and dress herself and to sit up in a wheel chair; for the last few weeks she has been able to stand and walk a few steps unassisted. On several occasions during her stay in the neurologic ward she has been observed to have attacks of rapid breathing, such as are often seen in epidemic encephalitis. On October 3, the patient complained of redness, pain and swelling of the right knee, for which she was given acetylsalicylic acid, 10 grains, three times a day. The pain has disappeared but the patient still complains of occasional stiffness.

The laboratory observations were: Spinal fluid, normal pressure, no globulin, 1 cell, negative Wassermann reaction and a flat gold curve. Blood chemistry showed a nonprotein nitrogen of 35, creatine 1.7, uric acid 2.5. The sugar tolerance showed: a fasting specimen 0.118 per cent, 0.133 at the end of the first hour, 0.105 at the end of the second hour, and 0.083 at the end of the third hour: the urine was free from sugar. The basal metabolism rate on June 30, 1927, showed minus 4, according to the Aub-Dubois tables and plus 9 according to the Harris-Benedict tables. On September 22, the basal metabolism rate was zero with the Aub-Dubois tables and plus 1 according to Harris-Benedict. A roentgenogram of the chest showed no evidence of tuberculosis and no mediastinal shadow. Roentgen-ray examination of the skull showed a sella turcica of average size and normal outline. A roentgenogram of the long bones showed no pathologic changes. The sputum was negative for tuberculosis. The electric reactions of the muscles were normal. Examination of the blood showed red blood cells 4,864,000, hemoglobin 88 per cent, white blood cells, 9,400, 68 per cent polymorphonuclear neutrophils and 32 per cent small lymphocytes. The results of the urinalysis were negative.

Physical examination at present, except for the remaining weakness and emaciation, gives essentially negative results. She is able to stand and walk a few steps unassisted and there is no evidence of any loss or diminution of automatic associated movements.

Comment.—This case was considered worthy of presentation because of the obscurity of its etiology, the rather unusual clinical picture and the fact that the patient survived a year of severe illness and lost at least 50 per cent of her body weight and yet is on the road to recovery. Loss of 40 per cent of body weight is as a rule fatal (Higgins, H. L.: Endocrinology and Metabolism, New York, D. Appleton & Company, vol. 4, p. 24, 1922). The following diseases have been considered as possible etiologic factors: Acute arthritis, complicated by epidemic encephalitis; acute rheumatic fever with resultant encephalitis.

My colleagues and I have considered that visceral pathology — endocarditis, nephritis and pulmonary tuberculosis — has been successfully ruled out. The possibility of a neurosis as a complicating factor in aggravating the severity of the symptoms must also be considered. Certainly she has the background of a neurosis, but this could scarcely explain the entire picture. The symptoms this patient shows — headache, delirium, insomnia, increased salivation, myoclonia and vomiting, with high temperature — all point to encephalitis. I

am unable to find reported any case of "epidemic encephalitis" showing such a picture of marked emaciation. Furthermore, there is the close association of this disease with acute, painful swelling of the joints, which may be an accidental complication of epidemic encephalitis, but it is certainly not usual. Delirium, high fever and choreiform movements have all been described among the nervous complications of acute rheumatic fever. The delirium and chorea are due to the involvement of the central nervous system during the course of acute rheumatic fever and can be explained on a basis of a toxic encephalitis accompanying this disease. I have been unable to find this symptom complex referred to as an encephalitis resulting from acute rheumatic fever, but the symptoms given above, which certainly are on an encephalitic basis, are commonly described as accompanying acute rheumatic fever.

Assuming that some type of encephalitis has been present in the patient, it is interesting to speculate as to the pathologic process or pathologic physiology necessary to produce the clinical picture. There is the possibility that this condition may result from an involvement of the sympathetic centers of the hypothalamus, considerable evidence of the existence of which has lately been brought forth (Kraus and Perkins: Syndrome of the Cerebral Origins of the Visceral Nervous System, ARCH. NEUROL. & PSYCHIAT. **18**:249 [Aug.] 1927). Conceivably, a disturbance of these centers might result in widespread nutritional disturbance. Next there is the possibility of an endocrine dyscrasia to account for this syndrome. The patient does not present the picture of a pituitary cachexia nor a cachexia thyreopriva. The classical syndrome of pluriglandular insufficiency of Claude and Gougerot, is described as invariably fatal. Necessarily, in such a widespread disturbance of nutrition as this, the endocrine glands must be affected. In view of the onset and course of this disease, and its varied symptomology, it is extremely doubtful that a glandular disturbance is the basic cause for the loss of weight.

Starvation is the most usual cause of emaciation. In this case there is a history of vomiting and nausea which have been more or less continuous over a period of a year. On admission there were symptoms of water starvation, such as dryness of the skin and scaliness of the scalp and lips, as well as general emaciation. Since the cessation of the nausea and vomiting, the patient has been able to take fluids and food and the condition has been rapidly improving. The history of high fever for the first four months of the illness, with its resulting increased metabolism, must have been another factor in the rapid loss of weight. The basis of the vomiting could well be an encephalitis. I am inclined to the diagnosis of acute rheumatic fever with a resultant encephalitis to explain the arthritis, headache, high fever, delirium, myoclonia, diplopia, tachypnea, insomnia and salivation which this patient has shown, and also the irritability of the vomiting center, which resulted in the virtual starvation of the patient.

DISCUSSION

DR. FOSTER KENNEDY: Dr. McKinney has asked us to assume that this patient has encephalitis. This is a large assumption. She may have had encephalitis but her symptoms might have been a part and parcel of any infection and it might not have been the result of an infection. Let us assume that she had a pancreatic defect, defective pancreatic function, intestinal indigestion in the great intestine — fat indigestion. I do not remember whether free fat was found in the stools or not. Can Dr. McKinney tell us that? Supposing she had a profound great intestine indigestion, she might easily have run into an emaciation such as existed when first seen, and her symptoms which gave rise to the idea that she had encephalitis might have been simply the result of an acidotic condition. I put that before you as a hypothesis as it is a case in which hypotheses are required.

DR. E. D. FRIEDMAN: It is my impression, as Dr. Kennedy has just stated, that this is an endocrine disturbance. If the history of the incidence of encephalitis be true, and it appears to be, one might speculate on the subject and assume that, possibly, instead of the usual sequelae of hypopituitarism

and obesity there is here the obverse of that picture, and it was a lesion of the tuber cinereum that gave rise to cachexia. The basal metabolism figures were never found much different from the normal, but such conditions have been described. Even in some cases of thyroid disease without evidence of active toxicosis, profound emaciation such as this patient presents has been encountered. I do not think we shall be far wrong in assuming that possibly both the endocrine and the vegetative nervous system are at fault.

Dr. T. K. Davis: I can only say that I am partial to the diagnosis of encephalitis and would put first epidemic encephalitis. At least the patient has had many of the symptoms of the encephalitic syndrome. She had excessive salivation, periods of diplopia, and periods of paroxysmal dyspnea. The last was not associated with acidosis or disturbances of consciousness. I believe that we do not know enough regarding endocrine disturbances after encephalitis to talk much about this point.

Dr. McKinney: As Dr. Kennedy said, this is a case in which the diagnosis is purely hypothetic. Visceral disturbances, including pancreatic disease, were considered as possible etiologic factors in this case. There was nothing in the history, physical examination or laboratory reports which led any who have examined her to make such a diagnosis. Visceral disturbances of sufficient severity to cause these symptoms have particular earmarks which this patient lacks. Of course there are diseases which can escape detection, but I am arguing on the basis of probability. The same reasoning applies to the diagnosis of pituitary cachexia. That also has certain pathognomonic features which are not present in this case. The blood chemistry is not disturbed, the sugar tolerance is normal, the basal metabolism rate is normal and the patient showed no symptoms of diabetes insipidus. While the diagnosis of pituitary cachexia is a possibility, it is not considered probable. The fact that the patient vomited over a long period of time and hence could not take nourishment, must be admitted as one cause for the loss of weight. There is practically no means of distinguishing between epidemic encephalitis and an encephalitis accompanying acute rheumatic fever.

A Visit to the Spanish School of Neuro-Histology. Dr. Lewis Stevenson.

Dr. Penfield has given American readers a short but excellent biography of Cajal and prefaced his article by the comment on Spain of Cajal himself which I shall repeat: "It has been said many a time that the problem of Spain is a problem of culture. It is necessary, in fact, if we would enroll ourselves with the civilized peoples, that we cultivate intensively the deserts of our land and brain, thus rescuing by prosperity and mental vigor all those national riches that have been lost in the sea and all those talents which have been lost in ignorance." Cajal's life has been spent in the solution of Spain's problem; how remarkably he has succeeded is slowly coming to light. Spain is awakening again and evidences of returning wealth and rapid progress against ignorance and superstition are everywhere apparent. There is an active interest among the intelligent people that will go far to increase educational opportunities of all kinds and scientific education in particular.

Although Cajal and his pupils have done so much to advance scientific knowledge, not only in Spain, but throughout the world, they have had to work under adverse circumstances and have suffered much from lack of money with which to buy books and equipment for their laboratories. Today Spain does not give the impression of a poor country—during my stay in Madrid the value of the peseta advanced steadily and wealth seemed more in evidence than poverty. So far, however, little of this material prosperity has found its way to the scientific laboratories.

When I reached the Instituto Cajal, Paseo de Atocha, No. 13, Dr. Cajal, who seemed glad to see me, at once provided a microscope and accessories and placed a table at my disposal next to that of Dr. Sanchez, a biologist renowned for his work on the nervous system of insects.

He commended me to the care of Dr. Castro and then hurried back to his library, where he spends most of his life. From time to time he emerged with a beautiful slide of the cerebellum, stained by his reduced silver method and illustrating perfectly the finer anatomy of the Purkinje cells, and the climbing and mossy fibers. One's first impression is that Cajal is a sad man and that he may be a little difficult of approach. Day after day, however, one is startled by Cajal's hearty laugh and will find him enjoying a joke. Throughout the day visitors come into the laboratory and, with a bow to Don Santiago, are soon in conversation with him. He discusses all sorts of things from a new set of serial sections of the rabbit's brain to the latest gossip about Primo de Rivera. Everybody worships him and daily, just before lunch, the entire personnel of the laboratory collects about him in the library to listen for an hour to his discussions. In order to "let us in on it," he repeated in French what he had just said in Spanish.

Cajal's method of teaching and thinking is objective — only if he sees a nerve fiber penetrating a mass of scar tissue is he satisfied that it can take place. If he cannot see the nerve fiber penetrating the scar, he does not believe that it is impossible until he has exhausted every method of his own and of all other workers who have discovered or worked with staining methods for neurofibrils. Some of Cajal's finest preparations are made by Golgi's method. Another principle to which he adheres is that of securing material for study immediately after the animal is dead, so that none of the cellular architecture found can be attributed to postmortem changes. He also insists that material be preserved in several different fixatives, the principal ones being formaldehyde, formaldehyde-bromide, alcohol, ammoniated alcohol, pyridine, aniline, and even weak solutions of silver nitrate.

Cajal is justly proud of his library. He lives and works in it and, with the aid of a secretary, writes his papers there, develops his photomicrographs in an adjoining dark room. The most interesting shelves in the library are those immediately behind the master's chair; they contain complete files of all the books he has written. The amount and the diversity of these are astonishing. Besides the two large volumes in French — "Histology of the Nervous System of Men and the Vertebrates" — are many works in Spanish quite unknown in America and England; they include works on general pathology, on anatomy, an autobiography, an important work on color photography and a volume called "Coffee House Chatter," which has nothing to do with neurohistology but is a collection of ideas about life in general and about Spanish life in particular. He has works on comparative neurology of the greatest interest, and particularly to be noted is a volume, written in collaboration with Dr. Sanchez, on the nervous system of insects. Cajal has always been interested in photography, and his stereoscopic plates in natural colors (Lumière) are beautiful.

The principal technical methods used in the laboratory are the reduced silver method for nerve cells, with blocks of tissue, and the gold sublimate method for neuroglia with frozen sections. In addition, the urano-formaldehyde method for glia and for Golgi apparatus is used. The Golgi method for the complete ramifications of the processes of the neuron is also much used. Several methods for myelin are in use, including those of Weigert-Pal, Kulschitsky-Pal and Spielmeyer. The Marchi method is no longer much used. Castro was using vital staining with methylene blue, especially on sympathetic ganglia.

Dr. Hortega's new laboratory is situated in the students' residence near the Hippodrome. Hortega is young and full of enthusiasm for his subject and for Spain. He loves Toledo only a little less than his work. Hortega's work during my stay with him was done entirely with frozen sections. The principal staining agent used was silver carbonate, made up with lithium carbonate for neuroglia, and with sodium carbonate for other tissues. For tumors and tissues other than the nervous system, the double impregnation method is used. Frozen sections are put into 2 per cent silver nitrate overnight and then, after washing, are heated in silver carbonate, reduced in formaldehyde and

toned in gold. Naturally, I spent a good deal of time on microglia, which was first described by Hortega. Oligodendroglia received its share of attention, and it is curious to note that in Madrid microglia is easier to demonstrate than oligodendroglia; the reverse is true here.

Hortega's laboratory is always busy; he has many visitors from all parts of Spain. Hortega is well known and has so many applicants for work in his laboratory that one table had to be occupied in shifts. With the help of a student, Costero, Hortega makes all of his own photomicrographs and lantern slides. This work is done during the night, between 10:30 p. m. and 2 or 3 a. m., in order to avoid interruptions and vibrations. Hortega's work is not confined to the nervous system; he has applied his silver carbonate stain to other tissues with remarkable success, and it is impossible to attend a medical meeting in Madrid without hearing the name of Hortega referred to over and over again in connection with work on the reticulo-endothelial system. He is modest and shy, quick and accurate in his movements, with a delightful sense of humor and a faculty of making one feel at ease. One gets the impression that there is a great force in Hortega, driving him to still more brilliant discoveries in neuropathology. He is known to his associates as Don Rio; they are obviously fond of him, yet aware of his greatness. The work done in Hortega's laboratory is now collected in the *Trabajos Del Laboratoria de Histopatologia de la Junta Pasa Amphacion de Estudios*, which is published periodically.

Resolution on the Death of Dr. Thomas W. Salmon

Deeply sensible of the profound loss sustained by the medical fraternity of New York through the death of Dr. Thomas W. Salmon, and mindful of the great significance of this loss for the psychiatric and neurologic group in particular, the members of the Section on Neurology and Psychiatry of the New York Academy of Medicine, in tribute to Doctor Salmon, unanimously adopted the following resolution at a meeting held on Oct. 11, 1927:

Resolved, That the appreciation prepared by Dr. Williams setting forth, as it does, an epitome of the many-sided and brilliant career of one of their most distinguished colleagues, together with a just estimate of his invaluable and epoch-making contributions to the field of medicine, neuropsychiatry and mental hygiene; and accurately reflecting, as it does, the sentiments of the members of the Section on Neurology and Psychiatry of the New York Academy of Medicine with regard to the high professional and personal esteem in which they held him, be, for the purpose of preservation in the archives of the Academy, incorporated in the records of this assembly, and be it further

Resolved, That a copy of this resolution be transmitted to the family of Doctor Salmon, with expressions of deepest sympathy in their personal bereavement, and in the hope that a knowledge of the love, admiration and high honor with which his associates in the medical profession regarded Doctor Salmon, already immortalized in the hearts and minds of all who knew him, may console them in a measure, however slight, in their great affliction.

SECTION ON NEUROLOGY AND PSYCHIATRY OF THE NEW YORK ACADEMY OF MEDICINE AND THE NEW YORK NEUROLOGICAL SOCIETY

Joint Meeting, Nov. 1, 1927

GEORGE H. KIRBY, M.D., *President of the New York Neurological Society, in the Chair*

ANEURYSM OF THE CEREBRAL VESSELS—CLINICAL AND PATHOLOGIC PRESENTATION. DR. IRVING J. SANDS.

Cerebral hemorrhage is common; one of its causes is rupture of intracranial aneurysms. Aneurysms of the cerebral vessels may be caused by: arteriosclerosis, septic or infectious emboli attacking the vessel wall and con-

genital weakness of the vessels. The symptoms, before rupture, are scarcely ever sufficiently characteristic to permit the making of a correct diagnosis. In the majority of cases, a diagnosis is made only after rupture has occurred. The symptoms of a ruptured intracranial aneurysm are: (1) those due to pressure on the surrounding structures of the brain; (2) those due to disease causing the aneurysm, i. e., arteriosclerosis, infectious endocarditis and other conditions and (3) those due to subarachnoid hemorrhage. The diseases which must be differentiated are: (1) encephalitis, (2) tumor of the brain, (3) meningitis and (4) sinusitis.

During the past year, I ventured a diagnosis of ruptured cerebral aneurysm in four patients. One of them had infectious endocarditis and died; autopsy was not obtained. The second patient was discharged from the hospital only ten days ago. The third patient is well and is able to do some work. The fourth patient died, and I was able to secure an autopsy. In this report, I shall present the last two cases.

Case 1.—A woman, aged 47, the mother of four children, suddenly experienced sharp pain over the right side of the head, especially over the right side of the forehead, and pain in the right eyeball, which was followed by stiffness of the neck and a temperature of 103 F. There were diplopia and slight drooping of the right eyelid. After four days, she was sent to the hospital with a diagnosis of meningitis. On admission, she was well developed and well nourished. The heart and lungs were normal. There was cervical rigidity and a bilateral Kernig sign. The pupils were unequal, the right larger than the left; the right did not react to light or in accommodation. The eyegrounds showed marked retinal sclerosis with many hemorrhagic areas; the veins were full and tortuous, the disc margins blurred. The right eyeball was turned upward and outward. There was marked tenderness in the right supra-orbital region, with hyperesthesia and hyperalgesia in the area of the right fifth cranial nerve. The abdominal reflexes were absent; the deep reflexes were diminished; no pathologic reflexes were elicited. The temperature was 104.2 F.; the respiration rate was 20, and the pulse rate 88. The blood pressure was from 170 to 180. A blood count showed 23,000 white cells with 82 per cent polymorphonuclears; the red cells numbered 4,600,000, and the hemoglobin content was 75 per cent. Urinalysis showed 2 plus acetone, but tests for sugar and albumin were negative. The blood Wassermann reaction was negative. The spinal fluid was bloody and under increased pressure; the Wassermann reaction was negative and no growth was obtained on culture. Examination of the sinuses did not show any evidence of suppuration or infection. Examination of the throat gave negative results. On the following day, while being examined, she suddenly had a convulsion, with contraction of the left upper extremity and a Babinski sign on the left. Death ensued in a few minutes.

Postmortem examination disclosed a tense, bulging dura on the right side. A large clot and free blood were found present in the right middle and posterior fossae when the dura was incised. The entire right hemisphere was covered with blood. A ruptured aneurysm of the posterior communicating artery of the circle of Willis was found on washing the brain. There was an organized clot on the right trigeminal nerve. There were numerous hemorrhagic areas in the tegmentum of the cephalic end of the pons and in the midbrain on section after formaldehyde fixation.

Case 2.—On July 2, a white, man, aged 33, a tailor, father of three children, suddenly experienced sharp pain in the left frontal region. He also had diplopia. On the following day, he had rigidity of the neck. He was sent to the hospital. On admission, he complained of headache, stiffness of the neck and occasional diplopia. On examination, he showed intact cranial nerves, normal reflexes, normal coordination and normal sensation. The pupils were equal and regular and reacted well to light, directly and consensually, and

in accommodation. The eyegrounds showed some blurring of the discs, more of the left than of the right. There was slight cervical rigidity and a slight Kernig sign. The spinal fluid was bloody and came out under 4 plus pressure. The fluid showed a negative Wassermann reaction, one plus globulin, and reduction of Fehling's solution after centrifugalization. On July 17, the fluid was again bloody. The urine showed a trace of albumin. The blood chemistry was normal. The blood count showed 7,000 white cells with 68 per cent polymorphonuclears; red cells 4,000,000; hemoglobin 70 per cent. The bleeding time was one and one-third minutes; the coagulation time 4 minutes. The blood pressure was 120 systolic and 80 diastolic. The temperature was 99.6 F.; the pulse rate, 54; the respiration rate, 18. The heart, lungs and abdominal viscera were apparently normal. The patient continued to complain of headache and diplopia.

On July 20, he showed marked cervical rigidity and a positive Kernig sign. The pupils were unequal, the left larger than the right. There was ptosis of the left eyelid. The left pupil was fixed; the right reacted sluggishly. There was blurring of the left eyeground. The right knee and ankle jerk tests were one plus; the corresponding reflexes on the left side were three plus. The abdominal reflexes were present.

On July 22, the left eye was closed; the left pupil was widely dilated and fixed and the left eyeball could not be turned upward or to the right. The corneal reflexes were normal. There was slight right facial weakness. A Babinski sign was present on the right. The deep reflexes on the right were still diminished. I then diagnosed the condition as one of ruptured cerebral aneurysm, probably of the left posterior communicating artery. The patient continued to improve and was finally discharged on August 2. At present he shows left-sided anosmia and a complete left oculomotor paralysis. He has headaches. However, he is getting along fairly well, and is able to work part of the day.

DISCUSSION

DR. WILLIAM STEINACH: On what clinical grounds did Dr. Sands make a diagnosis of aneurysm rather than of an ordinary cerebral hemorrhage? As far as I can see, the diagnosis was an inference from past cases rather than from any differential points.

DR. SANDS: What do you mean by an ordinary hemorrhage?

DR. STEINACH: Hemorrhage from an arteriosclerotic vessel. In the absence of symptoms of a neoplasm prior to the condition I do not think that a diagnosis of cerebral aneurysm is tenable. I wish Dr. Sands would give the points on which he made a diagnosis of aneurysm rather than of hemorrhage from an arteriosclerotic vessel.

DR. SANDS: Arteriosclerotic vessels in the brain do not rupture. May I refer you to the papers by Globus and Strauss?

In the first case, the woman suddenly was seized with severe headache, with a little fever, and a diagnosis of meningitis was made. A lumbar tap revealed bloody fluid. Leukocytosis and a rise of temperature will occur whenever a foreign body is present in any serous cavity. Blood is a foreign substance. The fever might have been due to the fact that the blood was present on the tuber cinereum. This deduction was discussed by Tilney and Casamajor in a paper on ventricular hemorrhage. The patient had arteriosclerosis and paralysis; the only other possible diagnosis, except aneurysm, was meningitis. This was excluded by culture of the spinal fluid and by smear. Encephalitis does not cause a fluid that is bloody to the degree found in this patient. Furthermore, the patient had marked retinal sclerosis. The left-sided Babinski sign made me think that she had a superior alternating paralysis. Had I reasoned more carefully, I would have said that the blood had trickled down and had reached the fifth nerve and the pons, as it had.

THE CLASSIFICATION OF EXTRADURAL SPINAL TUMORS WITH REMARKS ON THE ORIGIN OF THE SO-CALLED ENDOTHELIOMAS. DR. CHARLES A. ELSBERG.

Primary, secondary and metastatic extradural growths within the cranial cavity are relatively infrequent as compared with intradural tumors of the brain; it is rarely difficult to distinguish the one from the other. Extradural compression of the spinal cord by growths is observed in more than 25 per cent of spinal tumors. Most of the growths cause symptoms of the spinal cord or root by direct pressure; some, by interference with the blood supply and secondary myelomalacia; in others, the interference with function of the spinal cord is due to a sinking together of the bodies of several vertebrae and not to the direct pressure of the neoplasm.

The classification of extradural tumors without adequate differentiation between those that are primarily extradural and those that extend secondarily into the vertebral canal has led to considerable confusion as far as the symptoms are concerned. Extradural spinal tumors should be divided into: (1) primary extradural, (2) secondary extradural and (3) metastatic extradural.

In a series of 179 cases of spinal tumor (not including intramedullary or metastatic growths), forty-six were extradural. Of these, 52 per cent were primary and 48 per cent were secondary. The primary tumors arise from dura, nerve roots or epidural fat and blood vessels, or from the bony, cartilaginous or ligamentous walls, and are located entirely within the vertebral canal. Many of the primary extradural growths are benign, encapsulated and completely removable. Meningeal and perineural fibroblastoma, fibroma, angioma, and chondroma are usually single. Primary extradural chondroma occurs in two forms: the growth lies in the extradural space on the ventral or dorsal aspect of the dural sac, or it is derived from and firmly fixed to an intervertebral disc. Primary extradural sarcoma occurs in two forms: Either the growth is derived from the dura and does not become adherent to or involve the bony walls of the vertebral canal, or it begins within the canal, erodes the surrounding bone and grows outward, forming an hour-glass or dumb-bell shaped tumor.

The secondary extradural tumors are either benign or malignant, and extend into the vertebral canal through intervertebral foramina or by erosion of the bone. They arise from the vertebrae or paravertebral soft tissues and may be extensions of growths in the thorax or deep tissues of the neck or back. In these secondary growths, the true spinal symptoms begin only when the tumor has extended into the spinal canal or has interfered with the blood supply of part of the spinal cord. With some exceptions, symptoms of the spinal cord are more acute in onset and more rapidly progressive. In my series, the development of disturbances of the spinal cord was gradual in 23 per cent and rapidly progressive in 77 per cent.

Secondary extradural tumors should be kept distinct from the metastatic extradural growths. At least 30 per cent of patients with primary or secondary extradural tumors can be greatly improved or permanently cured. The proportion is larger in the primary than in the secondary, and greater in benign than in malignant growths.

With regard to the histologic classification of encapsulated tumors of the nervous system, I recommend the classification of Penfield: meningeal fibroblastoma (endothelioma, meningioma); arachnoid fibroblastoma; perineural fibroblastoma (solitary neurofibroma), and the neurofibroma of von Recklinghausen's disease.

The meningeal fibroblastomas, commonly known as endothelioma or meningioma, probably arise from cells of the perimedullary mesenchyme, which embryologically belong to the arachnoid, but which have remained with the cells of the dura or pia mater or in the arachnoid itself. I have found spinal endotheliomas in relation to every surface of the three membranes except underneath the pia mater. The study of the spinal endotheliomas has therefore contributed much to the understanding of the origin of this type of new growth.

(This paper will appear in full in *Surgery, Gynecology and Obstetrics.*)

DISCUSSION

DR. WILDER G. PENFIELD: The point Dr. Elsberg has made, and he is the first to make this statement that meningeal fibroblastomas are found both on the outside of the dura and on the inner surface of the dura without attachment to the arachnoid, is important. It was shown by Schmidt, and later by Mallory, that these tumors arise from arachnoid; for that reason, Mallory called them arachnoid fibroblastomas. Thanks to this evidence, it seems better to call them meningeal fibroblastomas, because they do not always arise from the arachnoid.

A further point of interest is that the location of these tumors brings some evidence against the conception of Burr and Harvey that the leptomeninges are of ectodermal origin, and favors the view of Weed and others who have maintained that the leptomeninges and the pachymeninges develop together as one structure. Both tumors are mesodermal. The facts that these tumors are made up of fibroblasts and that they form reticulin or precollagenous fibers and are yet derived from these meninges are further evidence that the pia-arachnoid is in reality mesodermal. However, none of this evidence can be taken as finally conclusive on so important a point.

DR. S. PHILIP GOODHART: Are there any marked histologic differences in the type of primary sarcoma to which Dr. Elsberg has referred? Their structure might be expected to be different, considering the difference in their manner of growth and clinical behavior, and since one form attacks bone and the other extends up and down within the vertebral canal. Is the latter type identical with sarcoma seen at the base of the brain which attacks cranial nerves?

DR. ELSBERG: There is much variation in the histologic pictures presented by sarcoma, and there is much to be learned concerning them. According to the evidence which Dr. Penfield and others have given, there is no essential difference between the sarcoma which has the tendency to extend along the dura within the vertebral canal without becoming adherent to bone, and the growth which remains localized perhaps over a few segments, surrounds the dura, but has a great tendency to grow outward into the extravertebral tissues, either through intervertebral foramina or by bone destruction.

CEREBRAL PNEUMOGRAPHY: DIAGNOSTIC AND THERAPEUTIC USE OF VENTRICULOGRAPHY AND ENCEPHALOGRAPHY. DR. WILDER PENFIELD.

Ventriculography by direct injection of air into the ventricles and encephalography by the spinal injection of air have come to be accepted diagnostic aids in neurology. The judicial use of these methods has been made difficult because of the lack of frank discussion of the advantages and dangers in the methods. Some workers have been dazzled by the brilliant diagnoses which can sometimes be made and have perhaps relied too greatly on the methods, minimizing the attendant dangers and forgetting that the diagnosis might sometimes have been made with equal definiteness by more careful neurologic examination. Other workers have been discouraged from further use of the methods because of a fatal accident or because of inability to interpret the pneumograms. It is necessary to use careful discrimination in regard to the type of case involved before forming a judgment concerning either the dangers or the value of cerebral pneumography by either method.

Pneumography in Diagnosis.—Both the spinal and ventricular injection of air for diagnostic purposes were first described by Dandy in a series of brilliant papers. Unfortunately, he failed to give explicit warning and technical advice concerning the method which might have helped the other neurologic surgeons and neurologists who have since found the method useful. The chief service of pneumography at present is localization of expanding lesions in the brain. When used for this purpose, as for other diagnostic purposes, the method must always be considered a supplement to other forms of neurologic examination, and when diagnosis can be made without its aid injection of air should not be employed.

The location of expanding lesions may be judged by alteration in the form of the ventricles. A ventricle may be obliterated by direct pressure, may be displaced away from the tumor or may be caused to dilate because of distal obstruction to the outflow of cerebrospinal fluid. It should be borne in mind that neoplasms and other expanding lesions push but do not pull on the ventricular walls. Thus, an expanding lesion in the right frontal lobe may cause obliteration of the right anterior horn of the lateral ventricle, narrowing of the right inferior horn of the lateral ventricle (so that this horn shows only as a narrow slit) and also dilatation of the body and of the inferior and posterior horns of the ventricle on the opposite side. It may likewise cause dilatation of the posterior horn on the same side. These enlargements are due to more or less complete obstruction of the ventricle at the foramina of Monro.

An expanding lesion on the lateral aspect of the hemisphere may partially obliterate the ventricle on that side and may displace both lateral ventricles toward the opposite side or down, depending on the position of the lesion. It is unnecessary to point out here the characteristics of the symmetrical dilatations which these ventricles undergo as the result of simple obstruction of the downward pathway of the spinal fluid into the posterior fossa and forward in the basal cisterns.

It is true that in certain situations, tumors may progress a long way, even to death, and give no evidence of their presence as seen by ventriculography. For example, an infiltrating tumor of the pons may cause death before giving rise to hydrocephalus, but such cases are rare, and with all the means at command the diagnosis of unlocalized brain tumor is as a general rule no longer justified.

In addition to the localization of an expanding lesion, pneumography may be used to rule out the presence of an expanding lesion and thus differentiate between a vascular lesion and an expanding one. If carefully done, air in the ventricles does not seem to give rise to an unwarrantable risk after an old cerebral thrombosis. Pneumography has been well used to localize the site of the block in hydrocephalus. Occasionally it has been found useful in localizing a block of the spinal fluid due to tumors of the spinal cord. Here, however, iodized oil 40 per cent offers certain advantages, the chief of these being that the slender column of air is much less readily seen through the thick tissues of the trunk than is iodized oil.

In addition to localizing obstructive and expanding lesions, pneumography is of help in the study of scars of the brain, and it should be borne in mind that a scar following direct injury to the brain produces alterations in the outlines of the ventricle in a manner different from that of an expanding lesion. Foerster has described what he called "wandering of the ventricle." This "wandering" takes place toward the side of the injury of the brain. Thus the whole ventriclar system may be pulled toward the site of the lesion, not pushed away from the site as in the case of an expanding lesion. Histologic study shows clearly the mechanism of this traction. Laceration of the brain and hemorrhage into the wound produce a connective tissue scar usually attached to the overlying covering of the brain. The great plexus of vessels which is found in such scars is pulled on, and this pull is transmitted directly to the cerebral vessels and from them to the brain substance by means of the perivascular attachments of astrocytes.

The choice between ventriculography and encephalography must be made according to the individual case. In general, encephalography permits a more complete study of the ventricles.

Among the untoward reactions are the rare hemorrhage from puncture of a tumor by a ventricular needle and marked subsequent increase of intracranial pressure. These reactions are seen in cases of tumor of the brain. The first may be guarded against by approaching the ventricle through the occipital lobes after examining the visual fields; the second, either by removal of the air after roentgenography or by immediate operation. The other

untoward reaction chiefly to be feared is sudden bulbar paralysis with evidences of vasomotor collapse. This may appear in encephalography in apparently normal persons when both the ventricle and the subarachnoid spaces are filled.

In a case of encephalography that I observed this state of collapse happened suddenly when the patient's brow was turned down and oscillated; I attributed it to rather sudden filling of the fourth ventricle with a large bubble of air. The patient's condition was serious for about ten minutes, after which respiration became normal and the pulse regular.

In general, encephalography may be used to advantage in the study of the subarachnoid space and cerebral convolutions in cases in which the intracranial pressure is not raised. It should rarely or never be used to study cerebral neoplasms. For this condition, ventriculography is less dangerous and more useful. The indications for cerebral pneumography must be carefully scanned, but the timorous attitude of some workers is no longer justified. The risk of the method in each case depends entirely on the nature of the lesion. In tumor of the brain, the mortality is much higher, but the necessity of localization is imperative.

Therapeutic Use of Lumbar Air Insufflation.—I wish to direct attention to the use of lumbar insufflation in the therapy of traumatic headache. Occasional observations of relief from chronic headache in the course of encephalographic studies have been made by Hirsch, Bingel, Foerster and Carpenter. Without knowing of these incidental reports of relief from headache, it has been my fortune during the past year to discover that patients with a certain type of headache may be treated by lumbar insufflation with almost invariable success as outlined in a forthcoming report. The first patient in the series was treated five years ago. He was suffering from severe frontal headache following a fracture of the skull six weeks previously. For purely diagnostic purposes, the air was caused to pass through the subarachnoid spaces to the site of the pain. To my surprise, the boy was completely relieved of headache and vomiting which had been his only complaints. Four years later, the expedient was again undertaken; this time for relief of headache and, as an experiment, to see whether a patient suffering from post-traumatic headache of eighteen months duration could be relieved. This patient's suffering had been so great that it was feared he would commit suicide.

Chronic meningeal headache has the following characteristics: Localized pain in the head and transient vertigo are the cardinal symptoms: The headache is invariably localized. It may spread to a certain extent, but is definitely referred to a particular part of the head. The pain is situated near the site of the blow in most cases, but there seems to be a tendency for the pain to be referred forward into the frontal region. In some cases, the pain is on the opposite side of the head from that which was struck. The pain is dull and is often described as "hammering." There are at times, in addition, fleeting, sharp, stabbing pains at the same site, which are more noticeable in bad weather. The headache is usually present every day, with some tendency to diurnal variation, often being worse in the late afternoon. Some patients are unable to sleep at night. The vertigo is usually present from the beginning. It comes on when the patient rises from a sitting position or may appear at any time and usually lasts for several minutes. There is no sense of rotation, but the patient finds it necessary to grasp something and hold on until the sensation has disappeared.

In general, there are no physical signs typical of the condition. The blow is often, but not necessarily, associated with a fracture of the skull. A period of unconsciousness following the accident has been usual but not invariable. There is a striking similarity in the patient's complaints which seems to justify the recognition of these complaints as a syndrome, and the uniform relief which lumbar air insufflation has offered indicates that the method, if properly used, may be specific treatment.

The pathologic change of the condition is not clear as yet. It has been frequently suggested by Foerster, Schwab and others that the cystic arachnitis serosa which may follow trauma to the head may be responsible for the post-traumatic complaints. The pneumograms of these patients have shown definite evidence of subarachnoid formation of cyst in two cases. In many of the cases, there is an escape of air from the subarachnoid space to the subdural space. In certain cases, roentgenography does not show any definite evidence of a pathologic process in the pia-arachnoid space. The number of cases investigated is too small to reach a conclusion with regard to the mechanism responsible for the production of the pain and dizziness. It seems likely, however, that the passage of the bubble of air through the subarachnoid space frees the brain so that fine adhesions are broken or a post-traumatic obliteration of the subarachnoid space is opened.

Lumbar air insufflation as used in such cases should be done with considerable caution and with all necessary safeguards. The treatments are done in the reclining position and movements of the head made so as to carry the air into the subarachnoid space, completely filling this space over one cerebral hemisphere. The portion filled should be the site of the pain and with little or no passage of air into the ventricle. The patient should be kept flat in bed until the air has been absorbed.

The suggestion is made that the same type of meningeal headache may possibly follow infection of the sinuses or mastoid region and may explain the chronic headache which sometimes continues independently of the primary infectious process. It is suggested that these inflammatory meningeal headaches may likewise be susceptible of relief by lumbar air insufflation. The method of insufflation should be used cautiously and never employed when there is already increased intracranial pressure due to a lesion in the posterior fossa.

DISCUSSION

Dr. Elsberg: This is an interesting suggestion and well worthy of further investigation. One must naturally be somewhat conservative — at least, I feel that way — in arriving at the conclusion that the injection of air into the subarachnoid space over the convexity will separate adhesions which have existed for a long period of time. Such adhesions must be localized so that the injected air can pass around and get to other portions of the subarachnoid space. However, the actual fact that relief was obtained by this procedure in a number of instances, stands for itself and is a matter of great interest.

Dr. Michael Osnato: I am extremely interested in this therapeutic method for the treatment of patients with post-traumatic headache. I feel, however, that it is wise to utter a word of warning, because of the great number of factors other than the actual structural damage that often enter into the situation in these cases: psychogenic, litigational factors. My suggestion would be to eliminate the types of post-traumatic headaches that are still concerned with litigation from this series. The rationale of the therapeutic method employed by Dr. Penfield appeals to me very much, in view of the pathologic processes that we know exist in these cases, which we have been accustomed to call "concussion" of the brain. They are not concussions; many of them are lacerations, and the lacerations occur chiefly in the tips of the frontal lobes, the tips of the temporal lobes, and in the temporosphenoidal lobe; they are superficial, often involving the meninges.

My experience has not been exactly that of Dr. Penfield.

These patients can seldom localize the headache. They generally complain of shifting sites. I can see that in a certain group in which there are demonstrable signs by encephalography of cyst formation that the headache may be localized; generally, however, the headaches are not.

The vertigo also is an interesting symptom. In a large series of these cases, I have made Bárány chair tests and in some cases caloric tests. In every case in which there was true vertigo, some changes were found as the

result of these tests. However, there was nothing definite; nothing was found that was the same in every case. Indeed, generally, merely hypersensitiveness to turning, with sharp vasomotor reactions, nausea and vomiting were the symptoms. These patients complain of vertigo after sudden changes of posture. It is common to see, after the patients perform the movements which they state bring on the vertigo, either nystagmoid jerks or a real nystagmus of the horizontal or rotary type which lasts for a varying time; at that moment, in the Romberg position, these patients exhibit unsteadiness. I regard these observations as confirmation of the symptom of dizziness or true vertigo, as all these patients complain of headache and dizziness, but not all have a true vertigo.

In the cases of localized headache, with the foregoing confirmation of the complaint of vertigo, such as I have described, I can see a possible indication for the type of treatment described by Dr. Penfield.

Dr. E. D. Friedman: Several weeks ago, I had the privilege of addressing this section of the Academy on the subject of encephalography. I do not wish to repeat the contents of that paper. While I agree with Dr. Penfield in part, I take issue with him on some points raised this evening. It is agreed that injecting air into the cavities of the brain is only an addition to diagnostic methods. It adds to skill in the localization of focal disease of the brain. I always try to make the clinical diagnosis first.

By means of the encephalographic procedure, I have been able to fill the ventricles from below and to obtain useful roentgenograms. The technic is practically the same in every case. The patient is taken to the roentgen-ray room immediately after the insufflation of air, and he remains in the same position until the first plates are made. I have been able to demonstrate air shadows in both lateral ventricles, except when there was a tumor mass in one hemisphere compressing the ventricle on the same side.

I do not think that the existence of increased intracranial pressure is a contraindication to this procedure. I have now carried out the test in about seventy-five cases, twenty-five of which proved to be tumors of the brain; in none of them have I observed any serious phenomena during or following the procedure. Instead of doing the patient harm, one may possibly relieve him to a certain extent by substituting a compressible gas for the inelastic cerebrospinal fluid in the subarachnoid space. It is true that the intracranial pressure at the end of the procedure in some cases has been found somewhat increased over what it was in the beginning, but this has been noted in only a few instances.

I believe that this method helps to localize neoplasms more accurately. I shall not go into detail at this time. In a few instances the surgeon has been given a little better clue as to where to direct his attack.

As Dr. Penfield has intimated, it is an invaluable aid in the differential diagnosis, often difficult, between vascular lesions and neoplastic processes. One frequently is confronted by this need for fine differentiation, particularly in cases in which there is a complicating hypertension with changes of the fundus and in which even the ophthalmologist is unable to decide whether the alterations in the eye grounds are incidental to hypertension or to choked disc. It is in this type of case and in those instances of focal disease without changes in the fundus that the method is extremely valuable.

I have never found it necessary to remove the air which is evenly distributed throughout the subarachnoid space and the ventricles. It seems to me that the introduction of air through a trephine orifice into the ventricles alone would disturb the pressure relations more than the uniform filling of all the cerebrospinal spaces with air. I also have observed phenomena of collapse in some cases, but they have never been serious. I have not had any fatalities, except in the case of a child with amaurotic idiocy.

The endolumbar insufflation of air also yielded therapeutic benefit in some cases of epilepsy. The convulsive seizures were reduced considerably, and

in two or three cases, the patient did not have attacks for a number of months. The Germans working in this field have also called attention to this fact.

I have found, further, that even if the ventricles are inadequately filled the first day, roentgenograms taken the day after reveal better air shadows. The air leaves the subarchnoid space after from twelve to twenty-four hours and finds its way into the ventricles of the brain.

It is my impression that this method has come to stay. I make this observation on the basis of the experience I have indicated. I believe it will not supersede ventriculography, but will supplement it. Trephine insufflation has its place in cases of tumor in the posterior fossa in which one would not deliberately attempt the lumbar method. It is also the method of choice in cases of chronic obstructive hydrocephalus. Occasionally one is unable to fill the ventricles by means of lumbar injection of air. In such instances, one may not infer that the ventricles are occluded and that the patient is suffering from internal hydrocephalus. In this type of case, the insufflation of air though a trephine opening has its place.

There are serious objections to the universal application of the trephination method. One has to plow through the brain, a procedure not to be undertaken lightly, as Dr. Penfield has proved. One occasionally induces hemorrhage from a vascular neoplasm. Often, the surgeon is unable to fill the ventricles adequately; the air often escapes. It is difficult to find the ventricles in case of their dislocation. By means of the lumbar method, all the air injected ultimately finds its way into the cerebral spaces, and one obtains insight not only into the shape of the ventricles, but also of the subarachnoid spaces and of the brain.

I have been able to demonstrate both the displacement of the ventricles in cases of tumor of the brain and the retractions with migration of the ventricles toward the side of the atrophic brain in cases of degenerative disease. I have obtained as much information with this method as is furnished by ventriculography and without any of the serious risks or disadvantages.

DR. PENFIELD: I find the same difficulty in understanding the pathologic changes that Dr. Elsberg expressed. Certainly, adhesions such as occur after infection could not be broken up by bubbles of air. It is conceivable, however, that when the bubble collects as it does into a mass of considerable size the displacement of the brain which must take place away from the dura, or possibly away from the arachnoid, may serve to break up very fine adhesions. It may be only the adhesion which follows the absorption of a blood clot in the subarachnoid space. I do not pretend to explain the pathologic changes; I only state the results which demand some sort of explanation.

As Dr. Osnato pointed out, in so-called traumatic neuroses, the headache may not be localized. Certainly, one would not suggest performing lumbar insufflation unless the headache were localized and unless the headache were of the type that I have described. The injection of air in cases of all kinds and types of headache would certainly be most unfortunate. In all except two of these cases the patients had litigation in mind or were receiving compensation of some sort. One man felt so sure of his relief that two weeks after leaving the hospital, he signed a release with the insurance company.

I was fortunate to hear Dr. Friedman present his paper two weeks ago and saw his excellent lantern slides. I think perhaps he misunderstood me. I did not suggest the removal of air after lumbar insufflation for encephalography. But in cases in which there is hydrocephalus, secondary to a tumor, and in which the air is introduced directly into the ventricle, the reaction can be reduced greatly by withdrawing the air immediately afterward and replacing it by the cerebrospinal fluid which has been saved in a covered dish for the purpose. I have thought that the disappearance of the air in the subarachnoid space was due to absorption there and that the increase in the amount of air in the ventricle that he found on the second day might perhaps be due to air which was in the spinal subarachnoid space at the time

the first roentgen rays were taken. Often the roentgen rays show that there is air in the spinal subarachnoid spaces, and as the patient lies in bed on his back the direct course for that air would be upward into the ventricle.

Neurologists have been slower in this country in taking up encephalography than they have in Germany. It seems to me fortunate that Dr. Friedman is taking it up and is doing such remarkable work with it. Perhaps the preference of neurosurgeons for the direct method of ventricular injection in cases of brain tumor is due to a certain extent to the fact that they can carry out both methods. In such cases, most neurosurgeons have considered ventriculography as a safer procedure than the injection of air by the lumbar route. However, we must defer to Dr. Friedman's experience in this regard.

THE MODERN TREATMENT OF TRIGEMINAL NEURALGIA. DR. BYRON STOOKEY.

In March, 1926, I read a paper on differential section of the trigeminal nerve for trigeminal neuralgia before the Section on Surgery and Neurology of the Medical Society of the State of New York. Up to the time of that report, Charles Frazier's subtotal resection of the trigeminal nerve, i. e., saving the ophthalmic fibers and motor root, was the procedure employed. The method suggested before the New York State Society was a further step in the refinement of the procedure which had previously been so brilliantly developed by Dr. Frazier, in that, according to the technic which I suggested, a true differential section of the dorsal root is now performed, selecting for section those fibers proximal to the ganglion which are derived either from the maxillary or from the mandibular division according to which division gave rise to pain.

This procedure has now been carried out for several years with signal success, especially in those cases in which pain has been limited to the mandibular division. Selection of the fibers derived from the maxillary division without injury to the fibers from the ophthalmic or mandibular divisions has been more difficult to perform and, as yet, has not been possible with uniformity since some of the adjacent fibers from the ophthalmic or mandibular division are likely to be cut in the attempt to segregate the fibers of the maxillary division from section. With further experience and greater skill in technic, it is believed that greater assurance may be developed and the auxiliary fibers identified as readily as are the mandibular. Differential section is especially desirable in those patients in whom the pain has been limited to the mandibular division. In these patients, the sensory fibers derived from this division can be cut and the motor fibers running with the mandibular division saved without injury to the maxillary fibers of the ophthalmic, thus destroying only those fibers along which pain has been carried without disturbing the motor division to the muscles of mastication and leaving the patient with a minimum anesthesia and its attendant paresthesia.

I called attention to the fact that separation of the trigeminal nerve into two nerves, the ophthalmic and the maxillomandibular nerve is well recognized in the comparative anatomy of this nerve, and that this separation of the gasserian ganglion into separate parts had been stressed by Tos, and by Frazier and Whitehead (Tos, Giglio: Anat. Anzeiger **21**:1900. Frazier, C. H., and Whitehead, E.: Brain **48**:458, 1925).

In addition, I pointed out that in herpes zoster the inflammatory process within the ganglion may be limited to the ophthalmic or maxillomandibular portions. Thus, suggestive clinical evidence is offered that a line of separation within the ganglion may exist as is indicated by its comparative anatomic development. Further clinical evidence of separation within the ganglion was pointed out in that pain may affect any one of the three divisions and that primary pain in the ophthalmic division is extremely rare.

(Patients were shown illustrating differential section as performed by Dr. Stookey during the past few years at the Neurological Institute, together with lantern slides of operations and additional cases.)

DISCUSSION

DR. S. E. JELLIFFE: I agree with what Dr. Stookey said regarding the barbarous character of alcoholic injections. Among the few patients whom I have referred to surgeons for this operation, for I consider it an operation and a serious one, I have never seen one who had received injections of alcohol who did not have a recurrence. The accidents that have followed in almost all the cases I have observed have often been worse than the disease itself.

I did not hear Dr. Stookey's complete paper, but I am impressed with the whole situation because this differential picking out of the fasciculi seems to me to be an ideal procedure. I should like to ask Dr. Stookey, if, in view of his experience, there are any electrical methods that might be applied to aid in the differentiation of the fasciculi, such electrical methods that would pick out the sympathetic reactions that may be going through the fibers, reactions that might be tested out before complete anesthesia is induced?

DR. STOOKEY: An electrode is used to pick out the motor fibers. The trouble is that considerable dispersion of the current occurs, especially when there is any cerebrospinal fluid in the wound, making accurate identification of the motor root by this method of little value. I do not believe the electrode would help to differentiate the sensory fibers. If it were possible to touch the different fibers and watch the patient carefully, I think one might find some help in reflex phenomena, which would be an aid as you suggest.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Nov. 17, 1927

JOHN FAVILL, M.D., *President, in the Chair*

QUINCKE'S EDEMA FOLLOWING PROSTATECTOMY. DR. D. M. OLKON.

This patient, aged 76, had had previous good health and habits and has not had a former similar complaint. Eight months before presentation, the prostate gland was removed to relieve urinary retention; the patient made an uneventful recovery. Three weeks after the operation, painless swellings began to appear on various parts of the body, at first of brief duration but later lasting for progressively longer periods until now they may remain several hours at a time. They appear on the lips, face, around the orbits, forehead, tongue, neck, hands, knees and scrotum; they are never painful, do not pit on pressure and are without fluctuation or redness.

Neurologic studies and serochemical examinations of the blood, urine and feces have all given negative results. The functions of the heart and kidney are good.

Many etiologic factors have been assigned for angioneurotic edema, but I have not found a report in which the prostate gland was given as a possible causative factor.

DISCUSSION

DR. MEYER SOLOMON: Was any serum injected?

DR. PETER BASSOE: Was any kind of irrigating fluid used in the bladder, or was any drug administered?

DR. OLKON (closing): Serum was not injected. I do not know what was used for irrigation of the bladder.

TWO SECIMENS OF PATHOLOGIC CHANGES IN THE PINEAL GLAND. DR. ROY R. GRINKER.

SPECIMEN 1.—This brain is from a young girl who developed a purulent ethmoiditis and subsequently a small abscess of the frontal lobe. Death took

place from generalized purulent meningitis. The general pathologist noted that although the child was only 12 years of age, the body appeared to be that of one at least 16. The pubic hair was of adult abundance but the breasts were not developed. The viscera were of adult size, the liver weighing 1,440 Gm. and the spleen 122; the configuration of the heart was similar to that in an adult. The specimen shows a small pinealoma measuring 5 by 3 mm. projecting over the anterior quadrigeminal body but not impinging on it.

SPECIMEN 2.—This brain is from a girl, aged 17 months, who died of bronchopneumonia. A cyst of the pineal body measuring 7 by 9 mm. is present. The walls are thin, and there is apparently no preserved pineal tissue. No pressure effects are to be observed.

Pineal tumors, although not rare, rarely cause the syndrome of macrogenitosomia praecox. Of all cases of this syndrome reported only one was noted in a female. Specimen 1 is from a rare example of such a syndrome in the female. The lack of development of the breast coincides with Müllers conception that the mammary gland is not correlated with secondary sex characteristics. The external configuration of the body and the size and weight of the organs were of adult type.

Evidence from the clinical side and from experimental feeding with the gland is at variance concerning the relation of the pineal gland in the endocrine system. The popular conception that pineal involution at puberty releases the activity of the sex glands is by no means proved. The cases I am reporting show the contradictions that pineal tissue was not present in a normal infant aged 17 months, while excessive pineal tissue was present in an adolescent girl with precocious somatic development.

EPIDURAL INJECTIONS OF NEOARSPHENAMINE IN THE TREATMENT OF PATIENTS WITH TABES DORSALIS. DR. GEORGE B. HASSIN.

In my studies of the pathologic changes in tabes dorsalis, I often encountered evident inflammatory phenomena in the epidural space, the dura, the perineural spaces of the spinal roots (especially the posterior) and the meninges. The inflammatory phenomena were associated with degenerative changes in the spinal cord typical of tabes dorsalis. This pathologic change suggested a method of treatment in this disease which consists in epidural injections of neoarsphenamine. The dose, 0.3 Gm. dissolved in 10 cc. of double distilled water, is injected with a spinal puncture needle through the hiatus sacralis into the epidural canal. The technic is the same as that introduced by Cathelin and extensively used in inducing so-called sacral anesthesia. The injections may be given once or twice a week for a period of four weeks or less depending on the patient's condition and the rapidity of improvement. They may be preceded by injecting 10 cc. of 2 per cent solution of procaine. To date, forty patients have been treated in the Cook County Hospital and the Research & Educational Hospital of the University of Illinois. They have received 125 injections. Almost every type of tabes was represented — that characterized by shooting pains, by gastric crises, by Charcot joints, by disturbances of the bladder, by optic atrophy and by other symptoms. Serious complications occurred in only one of the forty patients. This man, with a tabetic type of general paralysis developed phenomena of ascending paralysis and myelomalacia after the fourth injection. He died one month after the first injection. The necropsy revealed necrosis of the lowermost portion of the cauda equina and a softening of the sacral segments of the spinal cord. In one other case, signs of severe irritation of the cauda equina appeared with aggravation of the original signs and symptoms — ataxia, shooting pains, bladder incontinence and similar conditions. A considerable time elapsed before these symptoms subsided. To avoid such undesirable complications, one must not use force in introducing the needle and must turn the opening of the needle skyward. Against these two unfortunate results, I have given 125 injections without complications.

In the majority of cases improvement was marked and set in early, sometimes after the second injection. The most striking improvement was in locomotion, subsidence of the ataxia, reduction of shooting pains and improvement in the general physical and psychic condition. Improvement was not obtained in the optic atrophy or in Charcot joints; in gastric crises the results were unstable and inconstant. The serologic observations were influenced in some cases; in others they were not. Some patients have been given subsequent intravenous injections, and, strange to say, the condition became much worse. Chills would appear, accompanied by vomiting, nausea and shooting pains. Mercury rubs, however, given after a course of six or eight treatments, did not produce untoward complications. In general, marked and speedy improvement may be obtained by epidural injections of neoarsphenamine. This method, therefore, is worth a trial, at least in cases in which other methods have failed.

THE PATHOGENESIS OF THE ARGYLL ROBERTSON PHENOMENON, AND SOME OTHER SYMPTOMS IN TABES DORSALIS. SVEN INGVAR, University of Lund, Sweden.

The reflex immobilization of the pupil, or the Argyll Robertson phenomenon, belongs to the most important of the clinical symptoms, and is of equal importance to the ophthalmologist, the neurologist, the syphilologist and the internist. It occurs almost solely in syphilitic and so-called metasyphilitic diseases. Its occurrence in other organic nervous disorders must be considered exceptional, but in syphilitic diseases the symptom occurs in a rather typical form. It begins gradually and develops slowly to a miosis. There is generally an anisocoria, and, as a rule, the phenomenon is bilateral. In an attempt to explain the Argyll Robertson phenomenon, these characteristics of the syphilitic stiffness cannot be ignored. Many attempts to explain the pathophysiology of the phenomenon have been made, but all have failed. Knowledge of the afferent pathways of the pupillomotor reflex is not sufficient. The opinion of ophthalmologists in general is that the visual and pupillomotor impulses reach the nervous center by way of the visual nerve fibers, but many cases have been recorded, in which the patients have been thoroughly examined clinically, which cannot be explained by such an assumption. It must be assumed that there are two different sets of fibers within the optic nerves and the optic tracts: the visual and the pupillomotor. Bumke explains pupillary stiffness on the basis of degeneration of the collaterals of the visual fibers in the vicinity of the oculomotor nucleus; that cannot be demonstrated by the histologic methods available. Behr assumes an inflammatory focus in the crossing of the pupillomotor fibers immediately in front of the anterior quadrigeminal body as the cause of the phenomenon. In this way, he explains its customary bilaterality; the miosis is thought of as an irritation phenomenon from this focus, but such a focus has not been demonstrated anatomically.

For several years I have been occupied with anatomic studies of the diencephalon. Comparison of the thalamus of birds and mammals showed that the main morphologic pattern of the diencephalon is the same. This is of great theoretical interest, because birds and mammals show much marked discrepancies in the development of the cerebral hemispheres, and especially of the cerebral cortex. It shows the morphologic and the functional independence of the thalamus of the cerebral hemisphere.

In my studies, I was impressed by the fact that during evolution the phylogenetically oldest fiber systems of the diencephalon remain on the surface of this brain region, or are found inside in the vicinity of the third ventricle or the sylvian aqueduct. The morphologic behavior of the optic systems of the diencephalon is the most striking instance of this. The diencephalon which, with the infundibulum, can be considered as a funnel, is during the entire evolution embraced by the chiasm, the optic tracts and the lateral geniculate bodies. During evolution the optic tracts are stretched and twisted, the lateral geniculate bodies are rotated and in primates and man are turned almost

upside down; but they remain as superficial formations of the diencephalon. Also, within the chiasma and the optic tracts themselves, there are evidences that in the phylogenetically oldest system, the fibers from the periphery of the visual fields remain on the surface of the chiasm and of the optic tract, whereas the macular fibers, which phylogenetically are young, are found inside forming the most central regions of these same formations. This principle applies also to the posterior roots at their entrance to the spinal cord. They are here coated with a layer of unmyelinated fibers, first described by Ranson, and these unmyelinated fibers form the phylogenetically oldest fibers of the posterior roots.

My own studies have convinced me that there is a concentric laying down of fibers in the posterior roots. The phylogenetically youngest fibers run inside, within the roots, whereas the older fibers run closer to the surface. In the optic nerve, also, there is the same histologic evidence. It can be shown in the rabbit that in the posterior parts of the optic nerve, close to the chiasm, there is a surface layer of thinner fibers, which probably must be considered as phylogenetically older fibers within the optic nerve.

From these considerations I concluded that the afferent pupillomotor fibers must run on the surface of the chiasm and of the optic tract. From the experimental investigations of Karplus and Kreidl, it is known that the pupillomotor fibers take a surface route from the region of the lateral geniculate body to the anterior quadrigeminal body. According to Lenz, they probably cross in the most superficial regions of the posterior commissure.

Syphilitic diseases, especially tabes dorsalis, occur with chronic meningitic processes, which are particularly marked in the basal spaces of the brain and the diencephalon; these processes are known to cause marginal degenerations of these regions of the brain. It seems natural, therefore, to explain the pupillary disturbances that are so typical of syphilitic diseases as being due to such marginal degenerations. Thorough investigation of a case of early tabes dorsalis showed how the process gives almost exclusively concentric marginal degeneration of the chiasm and of the optic tract.

From the same point of view I investigated the course of the fibers from the levator palpebrae superioris and rectus superioris muscles, and found that in man, and especially in the macacus monkey, these fibers form a superficial layer in the stem of the oculomotor nerve in its course through the subarachnoidal space to the brainstem. These fibers are therefore more exposed than others in meningitic diseases. Syphilitic diseases also produce marginal degenerations of the oculomotor nerve stem, which explains the occurrence of the isolated ptosis in syphilitic meningitis and in tabes dorsalis.

In a previous paper I have shown that in early tabes dorsalis there is degeneration of the naked surface fibers of the posterior spinal roots. This is in accord with the observations of Lissauer that the marginal zone inside the spinal cord degenerates early in tabes. The marginal zone of Lissauer contains a great number of the naked fibers that originate from the posterior roots. The tabetic spinal symptoms are also evidence that the phylogenetically oldest fiber systems degenerate first in the early stages. The clinical symptomatology in early cases of tabes is dominated by disturbances or loss of phylogenetically old functions: loss of the tendon reflexes, of deep sensibility, of vibration sense, etc. In this way one can explain the spinal and the cranial symptoms in tabes dorsalis from the same uniform point of view, more satisfactorily than has been done before. With this explanation of the Argyll Robertson phenomenon, one can explain also all the clinical characteristics of the phenomenon. It is not necessary to refer to hypothetic foci; one can refer to the well known marginal degenerations in the basal regions of the brain in syphilitic diseases. It becomes intelligible that it must occur as an early clinical symptom; that it must be bilateral and why there is miosis and anisocoria. Other organic diseases of the brain seldom produce this symptom because they usually are not attended with degenerations in the marginal

region of the diencephalon. The slowly developing reflex immobilization of the pupils after traumatism of the skull can be explained as due to chronic meningitic changes of the basal spaces, caused by hemorrhage in the meninges.

DISCUSSION

DR. G. B. HASSIN: While the problems pertaining to the pathologic changes in the pathogenesis of tabes dorsalis are some of the most difficult in neuropathology, the one least understood is that concerning the Argyll Robertson pupil. Dr. Ingvar selected a complicated subject for his address this evening. The mere fact that numerous contributions exist on this particular subject denotes how little is known about the pathways of the light reflex, in normal as well as in pathologic states. I remember the eloquent lectures of Darkschewitch who tried to make clear to the junior students that the course of the pupillary fibers is by way of the pineal body and the posterior commissure to the anterior corpora quadrigemina. I also remember the articles of opposition by Bechterew and the contributions of Pawlow from van Gehuchten's laboratory. Yet none of these contributions, once considered so convincing, was even mentioned by Dr. Ingvar, probably because it was not worth while. The anatomic method is evidently not altogether appropriate, for even in the pathogenesis of lesions of the tabetic cord — a problem much simpler than that of the Argyll Robertson pupil — it has led so far only to wide dissensions. The subarachnoid space, to which Dr. Ingvar referred does play some rôle, though it is customary to consider it responsible for obscure pathologic conditions as little understood as the subarachnoid space itself. I came to the conclusion from my studies that not so much the subarachnoid as the epidural space is responsible for the root and other tabetic changes, but I am unable as yet to tell what bearing the epidural changes may have on the pathogenesis of the Argyll Robertson pupil. So far the clinical facts proved of greater value. In the dispensary of the University of Illinois years ago, I had an opportunity to see two cases of blindness resulting from complete tabetic optic atrophy; yet the pupillary reflex was present in both. On the other hand, countless tabetic patients show loss of this reflex with good vision, that is to say with visual fibers preserved. One must assume, therefore, that the optic nerve contains two sets of fibers — one visual and one pupillary. The exact course of the latter can hardly be definitely settled until it is possible to differentiate between the two sets of fibers under the microscope.

DR. SVEN INGVAR: The old theory about the coarser and finer nerve fibers of the optic nerve was developed by Gudden. According to him, the coarser fibers should be the pupillomotor, but this idea has been rejected as unsatisfactory for reasons that I cannot discuss here.

I agree with Dr. Hassin that there has been much inconclusive discussion in the literature about different sets of fibers for pupillomotor and visual purposes. I did not intend to suggest that it may be possible with available histologic methods to differentiate the pupillomotor pathways from the visual fibers. This, to my mind, is not necessary for a solution of the question of the pathophysiology of the Argyll Robertson phenomenon. The pupillomotor pathways run on the surface of the diencephalon for a long distance from the lateral geniculate body to the anterior quadrigeminal body. This is sufficient at present for my deductions. I have developed reasons for the assumption that they run on the surface in the chiasm. It is my conviction that a knowledge of the marked marginal lesions of the brain regions along the basal subarachnoidal spaces is important for an understanding of the Argyll Robertson phenomenon. This has not been proved, but as I consider the whole pathology of tabes and syphilitic meningitis, I think it is not premature to develop my theory. Of one thing I am certain — the explanation of the isolated ptosis in the early stages of syphilitic meningitis. This can be explained only according to the principles developed in my thesis. If I have been able really to convince you about this one thing, I shall be satisfied.

Book Reviews

DER MESKALINRAUSCH, SEINE GESCHICHTE UND ERSCHEINUNGSWEISE. By KURT BERINGER. Monograph of the Journal of Neurologie and Psychiatrie. Pp. 315, Berlin: Julius Springer, 1927.

In the past few years several interesting monographs have appeared discussing the history, the effects and the therapeutic value of meskal. It is surprising that such an ancient species of cactus should at this period command so much attention. Beringer discusses the subject in a most pleasing and interesting manner. The book is divided into two main parts; the first part takes up the history, physiologic effects and experimental results of meskal; and the second part consists of resumés of thirty-five case reports. Part I, is made up of twelve chapters, beginning with the earliest uses of the plant, its historic development, its numerous mystic and legendary significances gradually leading up to the experiments conducted at the Heidelberg Clinic.

The first chapters of the book are probably the most interesting, as they take one back to the early folk lore of the Mexican and North American Indians. It appears that, as early as 1692, a Spanish mission along the Rio Grande was designated by the name "El Santo Nombre de Jesus Peyotes"; the word Peyotes having reference to the cactus of this special region. As a rule the Indians called this plant meskal, while the term meskal buttons or meskal beans is frequently used. In 1886, Lewin procured some of the Mexican meskal buttons as he had learned much about their therapeutic value from the folk lore of the people. Somewhat later, Heffter isolated four alkaloids, mescalin being the most important and the one that is of special interest in this article. Spaeth has given meskal the complicated formula of tri-methoxyphenyl-beta-aminoethane. Lewin, who in a most painstaking manner went into the historical side of the subject, found that as early as between 1499 and 1596, a Spaniard named Sahagum, who lived in Mexico with Cortez and wrote on the wonders of the country, also described the effects of this plant. In following years other writers referred to the delirium produced by the use of meskal. It appears that the early uses of meskal were confined to religious ceremonies and other tribal rites. In the past fifty years the use of meskal has become more general and has extended into adjacent American Indian tribes.

The first authentic experimental work was carried out by Prentiss and Morgan in 1895. In 1896, Weir Mitchell used the drug and a year later Havelock Ellis described in detail his own experiences under the heading of "A New Artificial Paradise." The work of Beringer outlines the observations carried on by Stein and Mayer-Gross at the Heidelberg Clinic. The physical effects of the drug were carefully observed, but the most interesting phenomena were those associated with the changes in the special senses. The visual phenomena were most striking, as all possible types of visual illusions occurred. Color tone was greatly influenced; changes in perspective, and unusual distortions of all sensory impressions were especially marked. The effects of the drug did not confine themselves to visual changes alone as all special senses were affected to some degree. It is also interesting that under the influence of the drug, acoustic stimuli might precipitate optic sensations, thus, piano notes might precipitate a series of colors with the brighter or darker tones depending on the pitch of the note. From these experiments three basic reactions appeared to be present: (1) abnormal sense perception; (2) disturbances of consciousness, and (3) abnormal sensory phenomena. In the psychic sphere, disturbances in time, in thinking, variations in emotional tone and in the personality were noted. Some observers have likened the reactions to those produced by hashish and others to chronic cocainism.

In part 2, the experimental results in over thirty cases are fully described. Various mental phenomena are gone into in great detail and should form the basis of a thorough reference work for those interested in the physiologic effects of this drug. While the author suggests the possibility of its clinical use it is questionable if there is any field outside of scientific investigation to which it is applicable. All in all the monograph is readable, devoid of repetition and as a reference guide should find its place in any psychologic laboratory.

THE TIRED CHILD. By MAX SEHAM, M.D., and GRETE SEHAM, Ph.D. Price, $2. Pp. 358. Philadelphia: J. B. Lippincott Company, 1926.

One gets the impression from this book that the authors would classify the majority of behavior disorders of children in two groups: (1) those that occur in children with a congenitally defective nervous system who demonstrate their deficiency from birth, the nervous and asthenic types, and (2) those that occur as a result of chronic fatigue and commence after several normal years. It is with the latter group that this study deals.

The book is divided into three parts: (1) the fundamental principles underlying work and fatigue, (2) fatigue and associated factors and (3) the prevention and management of chronic fatigue. In the first part, the normal child is defined, and the methods of estimating normality are discussed. The authors point out that physiologic and psychologic development are related, and that any attempt to force the child into activities beyond its physical and mental development are bound to produce harm. In the second part, the symptoms of acute fatigue are considered. When similar symptoms of a more chronic character are found, the authors consider them the result of chronic fatigue. Chronic fatigue is produced by physical defects, improper activities and habits, and there may be an hereditary predisposition. The condition may be diagnosed by physical signs, but a functional diagnosis may be made also on the basis of replies by the teacher, parents and the child to a questionnaire concerning the child's activities. In the last section of the book, treatment and prevention are discussed. The former consists in the elimination of physical defects and the institution of rest, the latter in proper habit training and the suitable regulation of activities. The final chapter discusses briefly the influence of the environment and of various types of parents.

Throughout the book the emphasis is laid on chronic fatigue—a fatigue that results in behavior disorders and is caused by physical defects and improper habits. This may be true in a small number of cases, but one wonders if chronic fatigue in children does not bear a close resemblance to the exhaustive psychoneuroses of adults; namely, that both the fatigue and the behavior may not be symptoms produced by a complex emotional situation. If the emphasis in treatment is laid on the physical condition and improper habits alone, as little, if not less, can be expected in results as if the emphasis was laid only on the emotional situation. Moreover, such emphasis on the physical side would serve in many instances as another factor in increasing the emotional difficulties existing between the child and its parents, and thus would hinder effective treatment.

The book contains much valuable advice concerning habit training which should render it of great benefit to the intelligent parents of well adjusted children. Its perusual might be of value to those parents who tend to compensate for their own deprivations by overstimulating the accomplishments of their children. The numerous tables and schedules make it of value to the physician engaged in the management of children's problems, and the insistence on the necessity of recognizing physical conditions stresses a point of view that may be neglected somewhat in the maze of complexities that have combined to produce a behavior problem. It should not be forgotten, however, that the physical is only one aspect of the problem, and that the interaction of the emotional needs of the child, of its parents and of the other members of the environment is of equal, if not greater, importance.

LE SYNDROME PARALYTIQUE UNILATÉRALE GLOBAL DES NERFS CRANIENS. By RAYMOND GARCIN. Pp. 225. Paris: Legrand, 1927.

This thesis, from the service of Professor Guillain at the Salpêtrière, consists in a study of paralysis of the nerves at one side of the brain with particular emphasis on cases in which the involvement of the nerve is the most extensive. Thirty-eight instances are collected, of which seven are reported for the first time. The literature seems to have been covered thoroughly, and the clinical description of the cases is excellent. The etiology of the condition is usually neoplastic, but instances of basilar meningitis, and even of polyneuritis, are occasionally encountered. The tumor may be extracranial, but is usually of meningeal origin. Sometimes an infiltrating tumor of the brain stem will provoke widespread unilateral paralysis.

In addition to the complete unilateral paralyses mentioned, there are a number of partial paralyses, of which the author gives the following list: syndrome of the sphenoidal fissure, syndrome of the external wall of the cavernous sinus, syndrome of the petrosphenoidal cross roads, syndrome of the internal auditory meatus, syndrome of the posterior lacerated foramen, syndrome of the foraminocondylar region and syndrome of the posterior retroparotid space. Almost any tumor of the base of the brain will fit into one of these classifications.

The author objects to the use of personal names in the denomination of syndromes; almost any combination, and therefore an infinity of names, is possible; furthermore, one syndrome may be included in another (Schmidt in Avellis, Avellis in Jackson) and thus lead to confusion.

In a well marked case there is little difficulty in arriving at a diagnosis. In the early stages, there may be some doubt concerning the location as well as the etiology. In such cases, the roentgenogram may give considerable assistance. Stenvers' position for bringing out the profile of the petrous portion of the temporal bone and the internal auditory meatus is preferred. In conclusion, the author emphasizes the peculiarity of complete unilateral paralysis of the cranial nerves, making of it a syndrome which is characterized clinically by: (1) paralysis of all the cranial nerves on one side alone, (2) the absence of choked disk and signs of intracranial hypertension and (3) the absence of sensory or motor disturbances in the limbs. The only common lesion associated with this condition is basilar sarcoma, although syphilitic meningitis may be limited to one side and may produce widespread paralysis.